THE MECHANISM AND GRAPHIC REGISTRATION OF THE HEART BEAT

THOMAS LEWIS

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THE MECHANISM
AND
GRAPHIC REGISTRATION
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THE HEART BEAT.

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OF
THE HEART BEAT

BY

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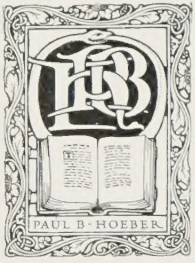

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PREFACE.

IT is now some eight years since I published, under the title of "The Mechanism of the Heart Beat," a book in which I attempted to review a collection of numerous, and at that time recent, researches upon normal and disordered action of the heart beat. The subject-matter at my disposal had been gathered by precise methods of research; it illustrated the application of such methods to clinical studies; it emphasised the value of reproducing in animal experiment changes witnessed during the progress of human maladies. For the last-named reason a number of clinical and experimental observations were placed side by side in the book so that they might be compared closely with each other. My general purpose was to show the solid progress which may be achieved by exacting methods and to preach an assiduous gathering together of all evidence in support of any given proposition. For it was in my mind, and still is, that clinical pathology, by which I mean the study of disease in the living man, suffers both from inexact observation and from hasty conclusion. Many methods of clinical observation, being subjective, lack accuracy. Some subjective methods are commonly believed to be effective only in the hands of a chosen few; such methods, while always open to scepticism, never can be productive. In no science, in the true sense of the word, is there the almost open vaunting of and reliance upon personal skill, in no science is there the veneration of supposed manipulative dexterity which we discover in our realm of medicine. Creditable methods of research are those which are capable of such

description or demonstration that the intelligent and diligent may employ them to the end results which their originators claim for them. It is by this test primarily that methods are weighed in science; it is by this test also that clinical methods will be judged eventually. A method which depends for its success upon a supposedly unusual sensitiveness of touch or of hearing, is a method whose clinical scope is admittedly limited, whose scope in science is almost negligible. Observations undertaken by any method which is not generally approved and which has not passed a rigorous censorship, are observations which add little to the sum of general knowledge. When we ponder upon the bedside tests, general and special, now in vogue, and ask ourselves how many of these have filtered through the sieve of rigidly controlled experiment, when we ask how many when filtering comes to them as come it must, will show a casting in so fine a mould that nets, though strictly meshed, will not entangle them; then do we probe the foundation upon which an art, aspiring to the place of science, rests. It is urgency—the immediate and daily promptings of the sick—which bolsters and preserves many of the expedients and opportunisms of the bedside worker. But these same expedients and opportunisms litter and obstruct the path of progressive knowledge. A physical sign or method, for lack of better, may serve the purpose of the moment, it serves no purpose in the pursuit of science till the measure of its fallibility is taken. Clinical observers, for the most part, do not yet recognise that many methods which they regularly employ, useful as they may be in the practice of an art, are inadmissible to the science. To advocate the general use of laboratory methods involving costly devices and time-robbing technique is not my desire; it is to emphasise the vital importance of methods of precision

in progressive studies of disease. While it is to be freely acknowledged that simpler methods are essential to the practice of medicine, it is clearly right to insist that in compiling reports contributory to scientific medicine precise methods are desirable. It is from this standpoint that the example of electrocardiography is to be stressed.

Inexact method of observation, as I believe, is one flaw in clinical pathology to-day. Prematurity of conclusion is another, and in part follows from the first; but in chief part an unusual craving and veneration for hypothesis, which besets the minds of most medical men, is responsible. Except in those sciences which deal with the intangible or with events of long past ages, no treatises are to be found in which hypothesis figures as it does in medical writings. The purity of a science is to be judged by the paucity of its recorded hypothesis. Hypothesis has its right place, it forms a working basis; but it is an acknowledged makeshift, and, as a final expression of opinion, an open confession of failure, or, at the best, of purpose unaccomplished. Hypothesis is the heart which no man with right purpose wears willingly upon his sleeve. He who vaunts his lady love, ere yet she is won, is apt to display himself as frivolous or his lady a wanton. For this reason oftentimes I was particular to set forth evidence upon evidence for a given conclusion in the book, feeling that no doctrine is sufficiently supported if yet another serious argument may be urged in its favour. For the same reason the leading theses chosen were those founded upon abundant evidence. My readers will find no reference to the still controversial question of the "neurogenic" or "myogenic" origin and transmission of the heart beat. The walls of the heart are composed of a syncytium of muscle fibres, closely interwoven with nerve fibrils and ganglia; in speaking of

the musculature of the heart, I have done so upon the distinct understanding that this term refers to muscle in full functional connection with the nerve elements surrounding it. For, dealt with in this manner, it is immaterial to the subjects considered and to the conclusions reached, whether one or other view is held.

It may not be inappropriate to refer here to a general terminology employed by writers upon disorders of the heart and especially to certain terms which are used to denote physiological properties of cardiac muscle. The strict separation of five cardiac functions, rhythmicity, excitability, contractility, conductivity, and tonicity, is one which, as I am well aware, is jealously guarded by some writers, more especially by the disciples of Engelmann. This doctrine may or may not be justified. Be that as it may, the classification of cardiac disorders upon this basis is neither exact nor serviceable. Therefore, in using the convenient terms *rhythmicity* and *conductivity*, and similar derivatives, they are employed only in so far as they indicate the observed facts, namely, the origin of heart beats in a limited area and in rhythmic fashion, the transmission of waves of contraction from one chamber to another, or from one portion of the musculature to another.

Physiological hypothesis does not concern pathologists; physiological observations do; pathological observations are still more relevant, and from these we may form an idea to guide us in our work. If the *basis* of our working hypothesis is still speculative, if physiological imagination governs it, as pathologists we work upon a foundation guaranteeing no reasonable security.

The nature of my subject necessitates abundant reference to graphic records, of which I have endeavoured to give examples in which the analysis will be unquestioned by those familiar with them.

Of the immediate value of graphic methods to practical medicine, it is my desire to speak but briefly. These records have placed the entire question of irregular or disordered mechanism of the human heart upon a rational basis, so giving to the worker the confidence of knowledge; they have influenced prognosis and have rendered it more exact; they have potentially abolished the promiscuous administration of cardiac poisons, and have clearly shown the lines which therapy should follow. The new clinical observations have stimulated and directed a host of laboratory researches, anatomical, physiological, pathological and pharmacological of a valuable nature.

The records in themselves constitute the most exact signs of cardiac affections which we possess. Imprinted by the disease itself, they form permanent and unquestionable testimony of events which have occurred, and may be placed in the balance, without disquietude, while experiences of a more subjective kind fill the opposing scale. Judged from this standpoint they possess also a great didactic value; they demand and impress accuracy of observation and, while nicely delimiting facts and fancies in their own sphere, sharpen the perceptions of similar boundaries in other spheres.

The book was written originally in the hope that it might stimulate the study of heart affections by precise methods. Its subject awakened a more widespread interest than I had anticipated. The exhaustion of the printed copies of what was intended as a monograph, four years ago, has forced me to consider the publication of a second edition. My objects remain unchanged, but eight years have brought many additions to our knowledge, it has hardened former conclusions, it has filled in many gaps. It has become possible and desirable to expand the scope of the book; but the

expansion, and some dissatisfaction with the original title, have persuaded me to change its name; while I have maintained the general plan of the "Mechanism of the Heart Beat" the text has been rewritten almost wholly and a new series of figures is employed, which I trust will be found clearer to decipher. A large proportion of the text is written as an abstract of my own observations; for this emphasis of my own work, I offer no apology, in that it seems to me desirable that an author should write mainly of that which has passed within the range of his own experience. Where I discuss the observations of others I have done so after fully studying their writings; when I have been in doubt as to the validity of experiments or the interpretation of them, I have, wherever it has been possible, repeated the experiments. Consequently, few phenomena are spoken of in this book with which I am not personally familiar.

To my old friend, Dr. T. Wardrop Griffith, of Leeds, I express my deep indebtedness for his great care in reading the proofs, and for the many improvements which he has suggested and of which I have fully availed myself.

THOMAS LEWIS,

September, 1919.

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Fig 1. A section (magnification, 330 diameters) taken through the *sulcus terminalis* of a pig's auricle and coloured with hæmatoxylin and Van Gieson's stain. The plane of the section is in the line of the sulcus and at right angles to the wall of the auricle. The epicardium lies to the left, the endocardium to the right. The muscle bundles of the auricular musculature are shown to the right, cut in their length and across. To the left, beneath the epicardium, is the dense collection of peculiar tissue of the sino-auricular node. It is compact, the muscle fibres are small and interlaced, and they present frequent nuclei. A large vessel is seen deep to this tissue, and numerous nerve fibres and ganglion cells are embedded in the node itself. (Published through the kindness of Dr. Ivy Mackenzie.)

Chapter I.

Special Anatomy.

The sino-auricular node.

Keith and Flack (*372*), while examining the musculature of the auricles, discovered a peculiar mass of tissue. It lies at the junction of the superior vena cava and right auricle.* (The position and extent of this node is illustrated in Fig. 2, 34, page 62, and 50, page 86.) "In the human heart, as in most mammalian hearts," these authors write, "an artery or arterial circle lies in the junction; the artery is surrounded by fibrous tissue in which are numerous peculiar muscle fibres, some nerve cells and some nerve fibres. The nerve cells and fibres we find from dissection to connect with the vagal and sympathetic nerve trunks."† "Our search for a well-differentiated system of fibres within the sinus, which might serve as a basis for the inception of the cardiac rhythm, has led us to attach importance to this peculiar musculature surrounding the artery at the sino-auricular junction." "In the human heart the fibres are striated, fusiform, with well-marked elongated nuclei, plexiform in arrangement, and embedded in densely packed connective tissue—in fact, of closely similar structure to the Knoten" (referring to the auriculo-ventricular node to be described presently). This description has been confirmed and extended by many workers (*383*, *385*, *387*, *692*). The special neuro-muscular system lies at the junction of the free border of the appendix with the superior caval termination, and extends downwards along the sulcus terminalis for a distance of about 2 cm. in man. In thickness it is approximately 2 mm.. The muscular fibres are small, being but a half or third the breadth of those of auricular fibres proper. This structure is termed the *sino-auricular node* (Fig. 1).

* The node lies, as Oppenheimer (*579*) has recently shown for the human heart, immediately to the venous side of the remnants of the old venous valves.

† Ganglia are scarce; the nerves break into "a plexus of moniliform fibrils in very close relation to its muscle fibres" according to the Oppenheimers (*580*). Argaud (7) describes the nerve supply as coming from a plexus situate at the root of the right coronary artery. The nodal artery is a branch of the right coronary vessel (see Fig. 2). Very beautiful pictures of the nerve endings in the *S-A* and *A-V* nodes have been published by Meicklejohn (*553*) more recently. According to this worker the nerve endings are highly specialised (in the monkey especially) and plexuses are formed around the *nuclei* of the muscle cells. (See also Eversbusch (*154*).)

As I shall show, the heart beat starts in this node, and the contraction travels from it over the walls of the auricle and reaches the structures described in the following paragraphs.

Fig. 2. A drawing to scale (nat. size) of the right auricle of a dog, seen from the right side. The preparation was hardened *in situ* and subsequently dehydrated and cleared with xylol. In this way the chief muscle bands and injected arteries of the auricle were displayed. The precise position of the sino-auricular node was ascertained by cutting serial sections through small blocks of tissue taken from the upper part of the sulcus terminalis. *S.V.C.* and *I.V.C.* = superior and inferior venae cavae. *P.V.* = right pulmonary veins. *S.A.N.* = sino-auricular node. The exact position of the node, its shape and extent vary in different animals within certain limits (see Fig. 34, page 62, and 50, page 86).

The auriculo-ventricular connection.

The anatomical connection between auricle and ventricle was discovered after Wooldridge (*793*) and Tigerstedt (*728*) had demonstrated functional continuity in the mammal, and subsequent to the elaborate researches of Gaskell (*215*), by which the dependence of the ventricular rhythm upon impulses derived from the auricle was clearly established in the cold-blooded heart.

The first account of muscular connections between the auricle and ventricle in the mammal, was given by Stanley Kent (*375*, *376*) in 1892. His preliminary paper was succeeded in 1893 by His's description (*317*) of a clean band of muscle fibres running from auricle to ventricle. In 1904 the foregoing observations were substantiated (*39*, *619*). The next advance in our knowledge was made by Tawara (*720*). In his book a complete account of the junctional tissues was given, and the anatomy of the whole system

and the connections with the network of Purkinje was described in great detail and in many species. These anatomical observations have been confirmed completely by more recent workers (*125*, *371*, *486*, *559*). Observers are agreed that a single path of anatomical communication exists between the upper and lower chambers of the heart (see second footnote, page 13).

The fibres of the junctional tissues may be traced from auricle to ventricle without break (Fig. 3). The system commences in the auricle in the neighbourhood of the coronary sinus and at the base of the auricular septum, where auricular fibres collect fanwise and interlacing unite with the *auriculo-ventricular node*. The node lies at the very edge of the auricular tissue at the posterior and right border of the septum. The bundle proper commences at the node, running almost horizontally forward and to the left, ensheathed and isolated in a canal,* and pursuing a course directly to the right of the central fibrous body of the heart to the *pars membranacea septi* of the ventricles. At the anterior part of this membrane, and a little in front of the anterior attachment of the median or septal segment of the tricuspid valve to the ring, the bundle forks. The left division of the bundle perforates the membrane, and still ensheathed lies upon the upper border of the muscular septum, and enters the subendocardial space of the left ventricle at a point immediately beneath the union of the anterior and right posterior cusps of the aortic valve. Its further course is downward, and it may be traced as it branches freely under the endocardium of the septum on the left side. The right division soon becomes subendocardial and, coursing downwards, enters the moderator band, or its representative, and proceeds directly to the papillary muscles where it breaks into its arborisation. In its course it marks the old separation between right ventricle proper and infundibulum. The arborisation of the left division (Fig. 4, 5 and 6) starts upon the septum, and passes to the papillary muscles of the mitral valve in two main branches. The arborisation on the right and left side is directly continuous with the extensive and complex subendocardial network of *Purkinje fibres*, which lines the greater part of the interior of both ventricles (Fig. 5, 6 and 7). From this network direct communication with the ventricular muscle fibres takes place. The smaller ramifications of the network frequently bridge the valleys between the muscular trabeculæ, and are there completely enwrapped by endocardium; these bridges are conspicuous at the apex of the left ventricle in almost every heart, be it human or not. It is said that the bundle and its branches are isolated by connective tissue sheaths beneath the endocardium until the papillary muscles are reached, and that no union takes place with the ventricular musculature during the earlier parts of the distribution; but this conclusion is based purely upon anatomical studies, and is not upheld by experiment (see page 125 footnote and context).

* Which Curran (*84*) regards as a bursa.

Fig. 3. A specimen in the Royal College of Surgeons Museum, photographed with the kind permission of Professor Keith. A human heart seen from the front and right. The anterior walls of the right ventricle and right auricle have been removed. The inter-auricular septum, the tricuspid valve, the papillary muscles (*G*), the moderator band (*F*) and interior of the infundibulum (*H*) are exposed. *A* lies in the right auricular appendix. *B* lies in the *ossa ovalis*. *E* is placed below the mouth of the coronary sinus; directly to the right of it in the figure an area of endocardium has been removed and the upper connection of the auriculo-ventricular node with the musculature of the septum has been exposed. It consists of a fan-shaped piece of muscle which lies directly below *D*. A bristle has been placed beneath the fan. From this point the auriculo-ventricular bundle and its right division are traced as they lie on a series of five bristles between *D* and *F*. The strand proceeds in a curved fashion to the membranous septum, which lies directly below *C*, and at this point the left division passes through the septum. The right division is continued upon the interventricular septum and enters and follows the moderator band (*F*) until it reaches the base of the large group of papillary muscles (*G*). The arborisation which commences at this point is not clear in the photograph.

Fig. 4. A specimen in the Royal College of Surgeons Museum, photographed with the kind permission of Professor Keith. The heart of a walrus dissected from the left side. The greater portions of wall of the left ventricle and left auricle (*A*) have been removed and the aorta has been divided vertically at its base (*J*) and the left half taken away. The interventricular septum and the cusps of the aortic valve are exposed. The anterior cusp of the valve is fully exposed and the mouth of the right coronary artery is seen. Directly beneath the posterior end (right hand end in the figure) of this segment, the left division of the auriculo-ventricular bundle enters the ventricle and immediately splits into two chief branches; these branches lie upon two horizontal bristles, over which there has been a very small amount of dissection. The further subdivision of the branches is perfectly clear, the arborisations are carried in free strands across the cavity; several large branches enter the papillary muscles, the bases of which are seen (*P*). Two long bristles are placed behind finer branches of the coarse network. *I* lies on the inferior cava; *G* on the pulmonary artery. Note the large collections of nerve tissue at the base of the heart; bristles are placed behind the thick strands at *G*, *H*, and *C*.

Fig. 5. (*Phil. Trans., 1915, B., CCVI. 181, Pl. 9*). A projected drawing (1½ nat. size) of the cavity of a dog's left ventricle, opened on the lateral wall between the papillary muscles. The overlying tracing shows the course of the left bundle division and its arborisation, as it was displayed by staining it for glycogen with alkaline carmine.

Fig. 6. ($\frac{1}{2}$ nat. size.) A colour photograph of the left ventricle of an ox's heart, opened to display the left division of the A-V bundle and its arborisation. The sheaths of these structures have been injected with India ink.

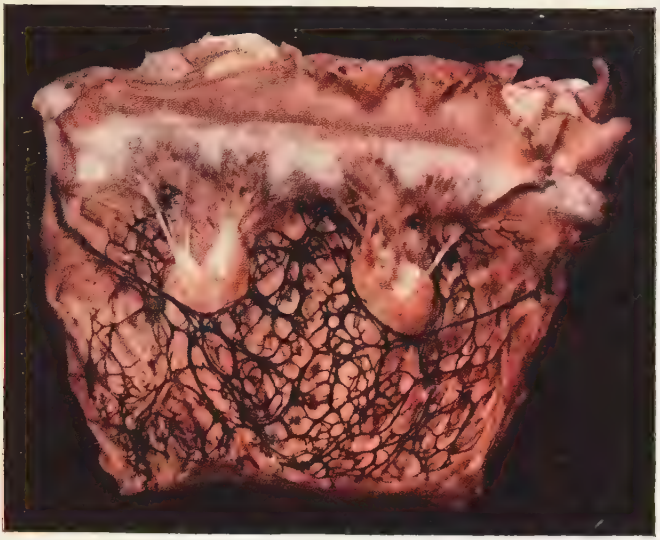


Fig. 7. ($\frac{2}{3}$ nat. size.) A colour photograph. The endocardial surface of the outer wall of the left ventricle in a sheep's heart. The sheaths surrounding the Purkinje strands have been injected with India ink. The picture is arranged to show the rich basketwork around the papillary muscles.

Fig. 8. A longitudinal section of the moderator band of a sheep's heart (magnification, 70 diam.). One of the free borders of the band is seen to the right (*En*, endocardium). Directly to the left of the endocardium is a number of longitudinally running and branching Purkinje fibres (*P.F.*). These are separated from the ventricular muscle (*V.M.*) of the band by a loose connective tissue sheath in which a small portion of a nerve (*N.*) is seen. Note the size of the Purkinje fibres and their large pale nuclei. (From a painting by Mrs. Aders Plimmer.)

The junctional tissues comprise :—

1. The auriculo-nodal junction or auricular node.*
2. The auriculo-ventricular node
3. The bundle proper.
4. The right and left divisions of the bundle.
5. The arborisations and the network of Purkinje.
6. The transitional fibres between network and ventricular substance.

The histological structure varies considerably from one species of animal to another and the most conspicuous differentiation of the tissues is seen in the hearts of ungulates, marsupials, and cetaceans (*768*). The following description applies particularly to the dog and to man, in which there is close similarity.

The auriculo-nodal junction (*53*) consists of fibres which are smaller than those of the auricle proper and which are arranged mainly in parallel fashion ; the transition to nodal tissue is abrupt, and here the tenuity of the individual fibre is remarkable. The *A-V* node consists of an intricate interlacement of the slender fibres, which cross and join at all angles. The fibres tend to be of spindle form and they are held apart by a rich network of connective tissue. Nerve fibrils and ganglionic cells are scattered profusely among them in many hearts (*165*, *553*, *786*), and there is usually a conspicuous arterial supply.

The fibres of the bundle proper have a more parallel arrangement (they are chiefly parallel in man) and are stouter ; they continue to increase in size as they are traced from bundle to arborisation and network, where they assume the proportions which in the ungulate heart are well known as characterising the fibres of Purkinje (Fig. 8). They appear swollen, striation is comparatively sparse, the nuclei are large, pale and frequently multiple. The vascular supply of these fibres is imperfect, and in many animals the fibres are surrounded by protective sheaths throughout their course. According to Tawara, the transition to the ventricular musculature is abrupt and consists in a rapid decrease in size with a corresponding increase in striation. The fibres of the bundle (*569*), and of the complete arborisation (*486*), have a rich content of glycogen which may be taken advantage of in displaying the system, for the glycogen may be stained with Best's alkaline carmine (*32*), (see Fig. 5).

Thus in the course from auricle to ventricle there is at first a diminution in fibre size (which is extreme at the *A-V* node), an increase in size (which is extreme in the network) and a subsequent and final decrease. The level at which transition to the Purkinje type occurs, and the degree in which this type is developed, varies much in different species ; the type is usually

* By some the " auricular node " and " auriculo-ventricular node " are regarded as distinct both morphologically and physiologically.

well represented in the fibres of the main branches, though it is less distinct in the human than in the ungulate heart.

The network of Purkinje and the two divisions of the bundle may be readily followed with the naked eye in the freshly opened ventricles of the sheep and calf: in the last named animals the sheaths of the system are well developed and, as these are readily injected,* the arborisation may be displayed to advantage (*59*, *491*), (Fig. 6 and 7); a short dissection reveals the bundle itself, and it may be traced with ease to the tissues of the auricle. The bundle in man and the sheep is from 1-2 mm. in thickness, and by the pallor of its fibres is conspicuous.

According to the modern view, it is by means of this bundle, and this bundle alone, that functional union between auricle and ventricle is affected; it is through this structure that the impulses from the auricle, which initiate ventricular contractions, are conveyed. The impulses must travel therefore from the neighbourhood of the coronary sinus to the right and left ventricles simultaneously. We shall examine this conclusion more closely in a subsequent chapter.

Morphology of the special tissues.

In the amphibian and fish's heart, as is well known, the veins enter the *sinus venosus*, a separate chamber separated from the auricular portion of the heart by a pair of venous valves.

In the mammalian heart sinus and auricle are fused, and morphologists have attempted to define the limits of the original sinus tissue by studying the relations of the entering veins. A strict separation of sinus and auricle is no longer possible in the mammalian heart. Keith (*372*) adopts the view that the greater part of the tissue of the auricles is an outgrowth from the primitive cardiac tube; the new tissue has spread over the sinus remnants, covering and displacing them. Tissue, which may be held to represent portions of the original sinus, is to be found in isolated masses at the superior cavo-auricular junction (especially in the neighbourhood of the *sulcus terminalis* and in the vicinity of the coronary sinus); these remnants I have already described as the sino-auricular and auriculo-ventricular nodes. Recent work, for which we are particularly indebted to Keith and Ivy Mackenzie (*373*, *400*, *401*, *496*, *497*, *498*), seems to show that the two nodes in question are not primitive remains, but rather special developments, representatives of which are to be found in all the lower vertebrates.

In the frog and eel, to take representative animals, a ring of specialised tissue exists around the bases of the venous valves; in reptilia and mammalia

* The first injections of this system appear to have been made by Sappey and described by him so long ago as 1853 in his "*Manuel d'anatomie descriptive*." He mistook the sheaths for lymphatic vessels. A historical account and beautiful pictures of the injected Purkinje and lymphatic systems will be found in Aagaard and Hall's monograph (*1*).

this ring is incomplete, becoming less complete as the scale is ascended; but what remains is still related to the original valves. The attachment of one valve is beneath the *sulcus terminalis* in the upper part of which the sino-auricular node is discovered. This structure, so it is considered, represents that portion of the original ring of tissue which lies in the neighbourhood of the great right vein, the right duct of Cuvier (now the *superior vena cava*). According to the same view, the auriculo-ventricular node or its junction with the auricle, represents a second remnant of the same ring, lying as it does in the neighbourhood of the great left vein, the left duct of Cuvier (now the coronary sinus), and within the original attachments of the same venous valves. The view that the two nodes are morphologically right- and left-sided structures is an attractive one and is supported by a good deal of evidence. It is not based solely upon morphological studies; the two nodes have a general structural similarity and are both intimately supplied by nerves; each is specially endowed with the power of producing rhythmic impulses, as we shall see; and lastly, the innervation appears to be, in a measure, homolateral, the right vagus and sympathetic nerve acting specially upon the region of the right or sino-auricular node, and the left sympathetic nerve especially upon the left or auriculo-ventricular node (see pages 164 and 191). This view also receives support from the recent work of Meek and Eyster (*549*, *552*), who find that the heart-beat of the tortoise and turtle starts, not as was formerly thought, in the great veins, but in the neighbourhood of the sino-auricular ring.

Again, in the frog and eel, a similar ring of tissue is found at the auriculo-ventricular junction and is prolonged in tubular form into the ventricle. As the scale rises, this ring also becomes incomplete and is prolonged to the ventricle over smaller reaches of its circumference. The auriculo-ventricular bundle as an isolated strand is peculiar to the mammalian heart, though in the monotreme *Echidna* (*373*) additional strands have been described.* Some remnants of peculiar tissue recently described by Kent (*377-380*), in man, are possibly derived from the same ring of tissue.†

According to Keith and Ivy Mackenzie, the nodes are portions of the original rings concentrated to form neuro-muscular contacts, being regarded by the former as analogous to the spindles of somatic muscle; the view is taken that during its phylogenetic development the heart tends to lose its automaticity and is brought more and more under the guidance of the central nervous system.

* In the mammal, and according to this view, the two rings or their remains have become united, the auriculo-ventricular node being continuous with the main stem of the bundle. A similar connection between the ring remnants is found in certain reptilia and fishes.

† There is no reason to believe that the structures described by Kent take part in conducting impulses from auricle to ventricle. The physiological evidence is strongly opposed to such a view, and such anatomical evidence as we at present possess is insufficient to give the view any material support.

ANATOMICAL NOTES.

Purkinje's fibres in the auricle. In 1909, A. G. Gibson described (*226*) certain fibres in the subendocardial region of the human heart, which he considered closely to resemble Purkinje fibres. These were found isolated and scattered over a considerable area, though especially in relation to the superior cava. His observations were published in the form of a preliminary communication, but have not been taken further.

In the same year Thörel published a preliminary communication (*726*) on a special muscular connection between the superior cava and auriculo-ventricular bundle in the human heart. In his full communication (*727*) he described an elaborate system of fibres, thought to be of similar nature to Purkinje fibres, running downwards towards the inferior cava from the superior cava and sino-auricular node. Thörel described these fibres as constant from heart to heart. But other workers have failed to find them (*384, 560*), and after examining Thörel's sections, attribute the appearances to pathological processes.

It is now generally held that no fibres comparable to Purkinje fibres are to be found in any part of the mammalian auricle.

Wenckebach's bundle. In 1907, Wenckebach (*761*) described a band of muscle fibres in the human heart, arising in the superior vena cava and coursing across the *sulcus terminalis* to the body of the auricle. An excellent figure of this bundle is to be found in Schönberg's monograph (*693*). Wenckebach, being in common with many workers at that time under the impression that the heart beat had its origin in the great veins, believed that this bundle might constitute a conducting path across the sulcus, from sinus to auricle. Following the suggestion of the same worker, that the condition we now recognise as auricular fibrillation might be a form of sino-auricular block, a number of pathologists, including Schönberg (*692, 693*), examined this bundle in cases which had shown gross irregularity clinically, and found in it widespread inflammatory lesions. Since the actual pacemaker has been isolated and the nature of clinical fibrillation has been recognised, this bundle of muscle fibres has lost its original prominence in the corresponding discussions.

Chapter II.

Mechanical Records of the Heart Beat with especial reference to intra-auricular pressure curves and venous records.

The subject matter of this book comprises in the main the analysis and explanation of disordered heart action. In studying these disorders, whether in experiment or at the bedside, we depend very largely upon recording devices of various kinds. Of such devices there are many now at our disposal. In the present chapter, which deals with mechanical as opposed to electrical instruments, I cannot hope to more than outline the methods employed and shall limit myself by describing those instruments, a general knowledge of which is necessary to the understanding of method, and a particular knowledge of which is needful for bedside observation. The chapter will also serve, it is hoped, to familiarise the general reader with the time-relations of the chief dynamic events in the cardiac cycle, thus introducing the more detailed studies of later chapters.

In studying the movements of the heart and vessels in laboratory experiment, several chief methods are employed. We may employ instruments which, attached to different parts of the muscle, record shortening of the fibres; we may use devices which record the change of volume in pulsating chambers or vessels; we may record the sounds produced by movement; lastly, we may employ instruments which record directly the changing pressures in the chambers or vessels. Each method has its separate purpose.

In animal experiment we are able to dissect and to display the actual organs studied; consequently our records may be taken directly from any desired part of the moving organ. But when we come to the bedside our task is more difficult, for we must then study the heart or vessels in the intact and uninjured body; we must study organs more or less deeply buried; consequently our methods are indirect and in general less exact.

Methods of investigation.

Curves of muscle shortening.—Many instruments have been used in taking these curves, which are named *myocardiograms*; for purposes of illustration I shall describe a single one in general terms. It is that devised

by Cushny (*88*); it has been used in obtaining the myocardiograms which illustrate this book. Two small rods of brass, perforated at their extremities, are attached to the desired cavity of the heart by means of single ligatures, so that the strip of muscle from which a record is desired lies between them. These two rods are united at their centres by a cross piece, to which the one rod is solidly attached and on which the other is nicely pivoted. When the muscle shortens, the pivoted rod moves towards the fixed rod at its muscular attachment, it moves away from the fixed rod at its free end. The movable rod is a lever and its free end drags upon a thread which, having passed under a pulley on the fixed rod, runs to a recording lever or other suitable device. The two rods, and the cross piece as a whole, are pivoted in such a way that this portion of the apparatus is free to swing with the heart. Thus the thread is pulled and the recording lever moves only when the muscle strip shortens, and the record is undisturbed by swaying of the whole heart or by movement transmitted from other parts of the cardiac tissue.

This myocardiograph is intended to record a complete curve of shortening and lengthening; but I use it only to signal the instant at which contraction begins, for in this respect alone it approaches accuracy. It is used in duplicate, one recorder being attached to the auricle and the other to the ventricle. The records are traced on smoked paper or photographed (see Fig. 26, page 50), and from them the beginnings of systole in the auricular and ventricular strips may be determined, while the sequence of chamber contraction is normal or disturbed, and their relations may be studied for many purposes with sufficient exactitude. Speaking with broad measurements in mind we may say that the curves tell us when systole begins in auricle and ventricle; speaking with fine measurements in mind this is not true, for all parts of the muscle in any given chamber do not contract simultaneously, neither is the error* of this instrument so minute as to be negligible when the beginning of contraction in the particular muscle strip investigated is considered.

Curves of heart volume.—In experiment, the changing volume of the heart is studied by enclosing this organ in a rigid box or *cardiometer*. When the whole heart is studied, it is usual to employ a glass receiver or bell, the wide mouth of which is slipped over the heart. The pericardium is tied short over the mouth of the bell, thus rendering it airtight. When the ventricles are alone studied, a rubber diaphragm is tied over the mouth of the bell and the heart is introduced through a central hole in this; the membrane sits snugly in the *A-V* groove and keeps the bell air-tight. A wide tube opens into the glass bell and communicates also with a recording tambour. When the heart contracts it draws air into the cardiometer from the tambour and

* Without exception all recording instruments yield an error; the rational use of an instrument depends upon knowing the degree of this error and upon determining that it is so small as to be immaterial in the circumstances for which the instrument is used.

the movements of the tambour-membrane are recorded. The tambour (or other suitable device) which is arranged to record volume, is so built that a large displacement of air may occur without any considerable alteration of internal pressure. Thus, a film of soapy water has been employed as the covering membrane of the tambour.

The volume curve of the ventricle signals with considerable accuracy the moment at which blood begins to leave the ventricle in its systole; it also indicates the moment at which the flow ceases.

Sound records.— A method of recording sounds is described in Chapter III. The method is applicable to man and animals equally. The beginning of the 1st heart sound and the beginning of the 2nd heart sound are used to signal the beginning and ending of the ventricular systole. When clear and sensitive records are obtained, these signals are amongst the most accurate which we possess in determining the times at which systole begins and ends; for, if they do not mark the closure, they mark the immediately subsequent tension of the auriculo-ventricular and semi-lunar valves, respectively.

Curves of pressure. In experiments on animals the changing pressures of the blood in the several cardiac chambers or in the vessels, are recorded by connecting the blood of these chambers or vessels to specially constructed recording instruments through rigid and fluid-containing tubes. In the case of the right auricle and the right ventricle, a hollow sound is introduced through the jugular vein until its end (open or guarded by a flaccid rubber bag) lies at the appropriate level; in the case of the aorta and left ventricle, the sound may be passed through the carotid artery. These methods of procedure obviate damage to the chest wall and the introduction of artificial respiration. To record the pressure changes in the left auricle, the chest must be opened and a cannula directly introduced through the auricular appendix or through a pulmonary vein. In some experiments, a cannula is introduced directly through the wall of either ventricle or of the right auricle. In taking pressures from the carotid or subclavian artery, the cannula is slipped within the end of the cut vessel and tied.

The sound or cannula, filled with a suitable fluid, is connected to the actual recorder, which also contains fluid, so that there is a complete column of fluid from the centre of the chamber or vessel up to the membrane which gives the record. In the earliest instruments this membrane had attached to it a lever or system of levers, which inscribed a curve on a revolving drum; but in taking modern records these levers are avoided and more sensitive and more accurate devices replace them. One of the best and most favoured of these is a tiny mirror, fastened to the membrane in such a way that it tilts when the membrane rises. A beam of light is then thrown upon the mirror and this beam is reflected and falls so as to give a spot of light upon a moving film.

Qualities of the recorder. I do not propose to describe the detailed construction of these instruments; those who desire such information may consult Wigger's recent and excellent account of them (*772*). But as accuracy of graphic records generally is intimately dependent upon the apparatus used, it is essential that those who use the graphic method should be familiar with the chief principles. Thus in pressure recorders the shape and size of the cannula or sound, the length, breadth and nature of the connecting tubing, the size, tension and sensitivity of the membrane, and the means by which its movements are translated into a graphic record, all influence, some more and some less, the actual form of the resultant curve. These factors have received for a long while close attention, and the pressure curves, regarded as representative, have altered in form as the apparatus has developed. The history of pressure curves is a history of instruments.

A chief quality of such an instrument is the natural *frequence* with which it oscillates. Every recorder has, like a fiddle string or pendulum, an oscillation frequence of its own, depending upon such factors as tension and mass.

If the natural frequence of the recorder is exceeded by the frequence of the oscillations which it is desired to record, these oscillations are damped or fail to appear in the curve. The tendency is always towards lightening the recording system and towards diminishing the size of the membrane, thus increasing its natural frequence. In modern instruments the weight of the moving parts is reduced as far as possible, whereby quickness of response is ensured and overswing, the result of momentum, is diminished. A high natural frequence entails small movements, consequently the actual movement is magnified by a system of optical projection.

Where the writing lever is substituted by a beam of light, the chief advantages gained are the reduction of weight and friction in the recording system without sacrificing magnification; but optical methods have a further advantage which deserves attention. A curve written upon a travelling surface combines pictorially two elements, the movement of the inscriber and time. In graphic records as we read them, time is represented (by the *abscissæ*) along a horizontal plane, the movement of the inscriber (by the *ordinates*) along a vertical plane. The form of the resultant curve is naturally dependent upon the speed with which the recording surface travels. It is also influenced, sometimes profoundly, by the natural line in which the inscriber moves. Almost all mechanical inscribers (for example all tambours fitted with simple levers) move through the arc of a circle, and this movement is expressed in and distorts the corresponding curves. When an optical system is used, it is so arranged as to draw, not a curved, but a straight line while the drum on which the record is written is at rest; while the drum is moving and the record is being inscribed, one source of curve-distortion is thus avoided. The optical system of projection has yet another advantage; when simultaneous records are taken, simultaneous

points in the several curves lie vertically above each other ; the photographic records of this book may be read in this fashion. The line records may not be so read, but simultaneous points are found only by referring to the corresponding index marks (see page 141).

Broadly speaking, the recording instruments of the modern laboratory belong to one class, the recorder moves in a straight line, its natural frequence is very rapid (being from 100-300 per second), and it is extremely sensitive. The instruments used to record pressure curves are so sensitive that they will depict even the fine ripples of pressure which constitute sound.

The instruments used for clinical records of arterial or venous pulse are of a different class, they are by comparison crude and will record only the chief pressure or volume changes and these in a distorted form. Records may be and have been obtained from patients with the sensitive instruments, but these cannot come into general use. They are fragile, they are intricate, they are costly and difficult to manipulate ; they are unsuited to the rough and tumble of routine bedside work. The crude recorder is the recorder of the practitioner. Its serviceability is demonstrated when it is stated that the sequence of chamber contraction can be shown clearly and sufficiently in a very high percentage of all cases by means of this simple clinical instrument. That is the chief information required, and it is not greatly added to by more sensitive devices. I do not mean to infer that the sensitive mechanical recorders will not yield additional knowledge ; they undoubtedly will. But in its proper field the crude instrument scores heavily by its ever-readiness, by the mass of material which it collects cheaply and in a short space of time. Coal when weighed for sale is not thrown into a chemical balance, neither is coal placed on a coarse scale when submitted to fine analysis. Both machines have their limitations Both classes of mechanical recorders have their limitations ; these should be recognised ; and according to the circumstances of the case, one or other is employed the more profitably. Supposing that in some unique case of irregularity the crude polygraph failed to decide the form of disorder, or supposing that the *form* of the several waves of the jugular pulse were to be the subject of inquiry ; then, a sensitive instrument would present decided advantages. But supposing that we desired to record the incidence of the chief forms of cardiac irregularity in particular diseases, or to study the influence of common irregularities upon the comfort or life history of our everyday patients ; then the sensitive tambour is extravagant for our purpose.

In describing mechanical methods of recording the human heart beat, therefore, I prefer a relatively crude method and relatively inaccurate curves ; I do so because these are the curves which are used, and which will continue to be used in analysing the sequence of chamber contraction in practice.

The venous pulse in animals and man.—Pulsation of the veins of the neck in pathological conditions is often such an obvious phenomenon that

it must have been recognised for many centuries. References to venous pulsation can indeed be traced in the writings of the early and middle years of the eighteenth century (*405*, *563*). In 1794, Hunter (*339*) described pulsation in the veins of a dog. Friedreich (*195*) took venous curves from the neck of pathological subjects in 1865; and two years later Potain (*610*) obtained simultaneous tracings of the apex beat, carotid, radial and jugular pulses from his sister; his description of the events and his interpretation of them are, in the light of our present knowledge, wonderfully accurate. From the time of Potain's contribution, observations have been published by many writers (*179*, *195*, *566*, *621*). In 1893-4, Mackenzie (*499*, *500*) published his first papers, which, with his collected observations appearing in 1902 (*501*), have given the impetus* to a careful investigation of the whole subject.

Pulsation in the veins of animals is not confined to the larger vessels feeding the auricles; it is a constant phenomenon in dogs, cats and rabbits, and may be seen extending in many of them into the smaller veins of the neck and limbs. Gottwalt (*232*) was of the opinion that the jugular veins of all normal persons pulsate, and this is now conceded. It is possible at the present time to obtain venous records from the neck of all normal individuals. The pulsation is usually large enough to be visible. Any agency which tends to promote a heightened venous pressure, such as raised intra-thoracic or abdominal pressure or gravitation, usually increases the force and visibility of venous pulsation. It is for this reason that tracings are taken most easily in the reclining posture, and that where pulsation is feeble, expiratory suspension of respiration increases its prominence. Occasionally, in pathological cases, the pulsation in the veins is very extensive. I have seen it in separate cases in the veins of the scalp, and in the veins of the forearm.

In man, the venous pulse is seen and recorded with the greatest facility in those veins which have but a short distance to travel before reaching the heart. Tracings may be taken direct from the external jugular vein when this is engorged, but more often the receiving instrument must be applied over the *jugular bulb*, as it is termed, which lies a little above and 25 mm. external to the sternal end of the clavicle (*370*). A needle passed back into the neck at this point strikes the internal jugular vein at a point where it is guarded by a pair of valves, which, when the path to the auricle is obstructed, or when blood regurgitates, produces a bulging in the vessel from which the jugular bulb derives its name Passed further on, the needle transfixes the subclavian artery (*370*). Tracings are obtained from the neck, in which the sternomastoid is relaxed, by applying to it with light pressure, a small receiver, whose width is about 4 cm. and depth 1 cm.. The interior of the

* Mackenzie's work was quickly followed by a large series of papers, in which the origin of the chief waves of the venous curves were discussed in detail; the following papers may be cited (*13*, *17-19*, *94*, *157*, *429*, *565*, *632*, *760*).

shallow cup communicates by air transmission with a tambour carrying a writing style. This simple apparatus is the most satisfactory as yet invented for general practical work. The curves obtained from the jugular bulb are frequently complicated by the primary wave of the arterial pulse, which in clinical work is not without its advantages, and by the respiratory movements when these are present. Curves are best obtained from the right side of the neck, for here the course of the innominate is shorter from neck to heart; clinically they may sometimes be taken from the pulsating liver also.

With regard to the rest of the mechanism little comment is necessary; the apparatus described may be adjusted to a modified sphygmograph, as in Mackenzie's original instrument, and the whole fitted with a reliable time-marker. Another and convenient arrangement for clinical work is the ink polygraph (Fig. 9), with which tracings of any length and at various speeds may be taken. It allows a simultaneous record of any two pulsations, and carries a reliable time-marker.

The natural frequence of clinical recorders is about 5-20 per second. They are not sensitive and, on account of their weight, the levers over-ride the mark, exaggerating the height of the peaks and frequently showing purely artificial secondary oscillations on the downstroke. Only the major waves are recorded, these are given unnaturally rounded outlines, and finer waves are slurred over or combined. Despite these defects, the instrument, when properly employed, is a most valuable one.

In animals, curves have been secured from the wall of the vein or blood stream by many workers, using very similar apparatus (*179*, *185*, *232*, *259*, *565*, *616*, *632*).

Latterly, more delicate instruments have been used both in man and animals and these have yielded more accurate curves than those we formerly possessed (*81*, *82*, *105*, *548*, *601*, *739*, *774*, *801*).

The venous record explores chiefly the movement of the right auricle.

Oesophageal curves.—If a hollow sound carrying a small rubber bag at its extremity is introduced into the œsophagus and this bag is distended and the sound connected to a recording tambour, curves may be obtained which throw some light on the movements of the left auricle. Such curves have been studied both in man and animals (*52*, *346-349*, *352*, *353*, *429*, *557*, *558*, *614-616*, *796*).

The method is clearly impractical as a routine clinical method, but it has served to show in specific instances of irregular heart action that right and left auricles participate equally in the disorder.

Cardiographic curves.—While venous records indicate the movements of the right auricle, and œsophageal curves may be used in special cases to signal movements of the left auricle, curves taken from the heart's maximal impulse or from a pulsating epigastrium, may tell us when the left and right ventricles respectively enter systole. The curves are taken by applying the same shallow cup as is used for venous work, though with

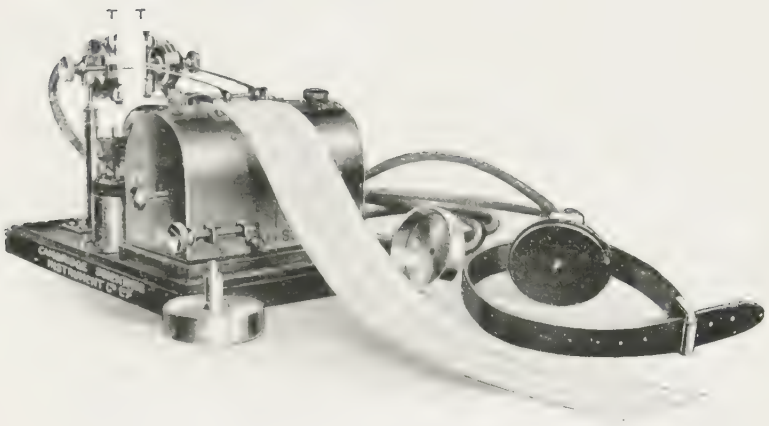


Fig. 9. The author's polygraph is a modification of the original ink polygraph of Mackenzie (*514*). The clocks which drive the paper and (one-fifth second) time-marker are encased and fixed to a wooden base. The speed of the drive is altered by means of the screw *S*; the key (*M*) winds the time-marker. Behind the clock are the two transmitting tambours (*T*, *T*), nicely pivoted on upright bars by means of universal joints. The tambours are smaller than in the original patterns of polygraph, and the air space within them is much reduced, so as to give greater speed of movement and more accuracy. The ink-writing pens, which project to the writing platform, are of an improved type. The reserve paper is carried on a drum (*D*) beneath the tambours, thereby space is economised and the travelling paper moves away from, and not towards the writing points. The transmitting tambours are connected through moderately thick-walled rubber tubing to the receiving apparatus. The venous and apical receivers (*R*, *R*) are simple cups; the radial (or brachial) receiver is a glycerine pelotte (*P*), fixed to the arm by a leather strap and having a fine tension adjustment attached to it. The ink bottle (*I*) is fastened to the wooden stand.

In the portable form of this polygraph the wooden base forms the bottom of the case in which it is carried with extra rolls of paper and spare pens. The polygraph stands ready for use when the case cover is removed.

The polygraph as a whole has been designed to combine strength with compactness, and is arranged so that as little time as possible is lost in packing it up, or in opening it out for use. It is made by the Cambridge Scientific Instrument Company.

firmer pressure, to the pulsating area. Of *mechanical* curves the cardiographic yield the most accurate signals of the beginning of ventricular systole; nevertheless they are subject to considerable error. Indications of the corresponding auricular contractions are frequently seen upon these curves, for, when the auricle empties itself into the ventricle, it transmits a wave through the ventricle to the recording tambour. Cardiographic curves are not widely used to-day in the analysis of disordered heart action.

Carotid and radial curves.—The usual standards, from which the venous curves are analysed, are arterial curves, taken from the radial, brachial or carotid artery. Clinical polygraphs are provided with an arterial receiving tambour, one example of which is shown in Fig. 9. It is secured over the artery and connected to the recording tambour by rubber tubing. The arterial curves are intended chiefly to signal the upstroke of the pulse, which is used as a standard of measurement; they are not intended to portray the form of the arterial pulse, though indications of the dicrotic wave are frequently to be seen in the curves. They are crude curves. But like other crude curves, they are crude in a particular sense. To illustrate this point, it may be remarked that the interval between the arterial upstroke and the dicrotic notch is, in these curves, a very inexact measure of systole in the arteries; on the other hand the interval between two arterial upstrokes is not inexact; it is almost if not quite as exact as in curves taken with far more delicate apparatus, and this is so because the error in each upstroke is the same. As it is the interval between pulse beats which is utilised almost exclusively in measurement, these curves satisfy our requirements.

The records and their meaning.

In Fig. 10 a series of curves is drawn to show the relations to the chief events of the cardiac cycle. The figure purports to show the curves of the human heart; but, seeing that several of the curves are not to be obtained from the human being, it is in reality a composite picture deduced from such human material as we possess, and supplemented by curves taken in animal experiments. Thus the electrocardiographic curve and the curve of heart sounds are actually human and are as exact in their detail and time relations as are any curves taken in animal experiment. The carotid and jugular curves are represented in the form of crude clinical curves; the relation of these curves to each other and to the remaining human curves are expressed without material error. The form of the remaining curves (aortic, ventricular and auricular pressure curves) and their time-relations are based on animal experiment (*598-600, 711*), but the curves have been modified so as to render them compatible with the duration of the human cardiac cycle.

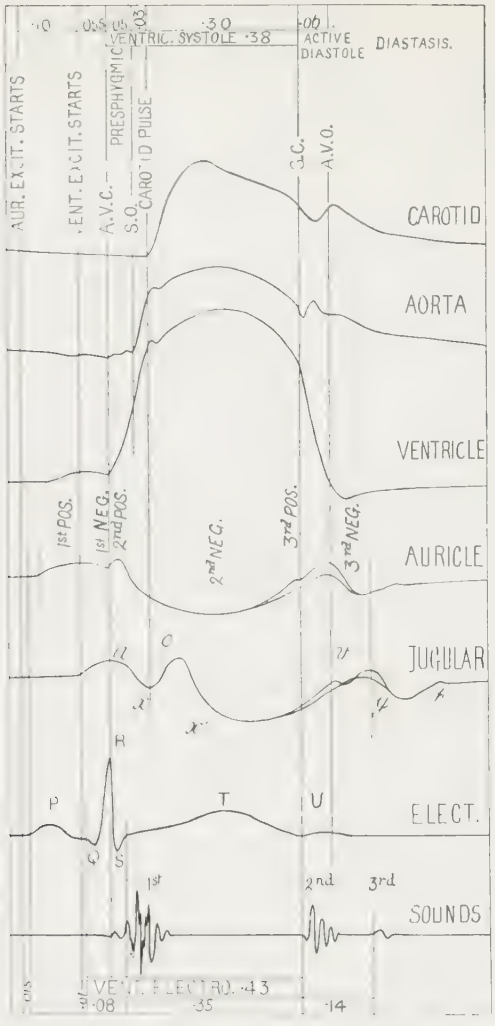


Fig. 10. A diagram intended to represent the curves of pressure in the carotid, aorta, left ventricle and right auricle of the human subject; showing the supposed or ascertained time-relations of these curves, the jugular pulse curve, the electrocardiogram and apical heart sounds to each other. The diagram has been constructed so far as possible from curves taken from man, but where such are non-existent the diagram has been completed by curves obtained from the dog, these curves being somewhat modified in their time-relation to correspond with such information as is to be obtained from the human subject. In constructing this diagram I have used my own curves as a basis in most instances, but have checked the time-relations of different events from the published curves and figures of other writers; attempting in so doing to arrive at a compromise where, in matters of detail, different observers have varied in their findings.

The times at which the excitation wave begins in auricle and ventricle are represented by vertical lines; the movements of the valves are also represented in a similar way. The scale of abscissæ is 1 mm. = 0·015 sec..

A.V.C. and *A.V.O.* = closure and opening of auriculo-ventricular valves, respectively; *S.O.* and *S.C.* = opening and closure of semi-lunar valves, respectively. Other verticals are drawn at convenient points and the chief time intervals are marked above and below in seconds.

The intraventricular and aortic pressure curves.—In the first part of the ventricular curve the end of diastole is shown; it is interrupted, immediately before the steep rise of pressure, by a low elevation which is due to the contraction of the auricle, whereby the pressure in the ventricle is somewhat raised. A corresponding wave is to be seen on the curves of aortic pressure. The chief rise of intraventricular pressure begins on the line marked *A.V.C.*, a line which represents the closure and tension of the auriculo-ventricular valves and coincides with the beginning of the first heart sound; here is the *beginning of ventricular systole*. The rise of aortic pressure begins later, on the line marked *S.O.*; this line marks the opening of the semi-lunar valves. The space between the closure of the auriculo-ventricular valves and the opening of the semi-lunar valves has an approximate length of 0·05 seconds; it corresponds to the period during which the ventricular pressure is rising, but during which it does not mount above the pressure in the aorta. No blood leaves the ventricle during this phase, which is spoken of as the *presphygmic period*; but the movement affects the aortic pressure slightly and the aortic pressure curve shows in this period one or more minute waves. At the opening of the semi-lunar valves the pressures in ventricle and aorta are almost the same; they continue to be alike and rise to summits, and are prolonged into the rounded plateaux which follow. During the whole of this period the blood is leaving the ventricle for the aorta and leaving the aorta for the periphery. Towards the end of the plateau stage the pressures are falling and, in the neighbourhood of the line marked *S.C.*, the pressure in the ventricle begins to fall away rapidly; it falls below that prevailing in the aorta, as the semi-lunar valves close and come into tension. This instant of the semi-lunar valve closure and tension (line *S.C.*) corresponds to the beginning of the second heart sound, and is marked in the aortic curve by the dicrotic incisure; here is the *beginning of ventricular diastole*. The ventricular curve now falls steeply as the muscle of the ventricle relaxes; the aortic pressure curve is meanwhile marked by the dicrotic wave, and the pressure curve then falls gradually as the arterial blood is gradually squeezed out into the veins. The fall of intraventricular pressure is continued past the line marked *A.V.O.*, a line which marks the opening of the auriculo-ventricular valves. These valves open when the ventricular pressure has fallen so low that the intra-auricular pressure exceeds it. The interval between the closure of the semi-lunar valves and opening of the auriculo-ventricular valve approximates to a twentieth of a second in man. The ventricular pressure continues to fall after the opening of the valves, but soon it rises as the chamber fills from the auricle; the chamber fills quickly during the early phase of diastole (active diastole), it fills less quickly during the later phase of diastole, a period spoken of in slow acting hearts as the period of *diastasis*.

The carotid pressure curve.—The curve used in the diagram is the crude clinical curve taken at the root of the neck. The rise of pressure starts

somewhat later than the rise in the aorta; the interval is approximately 0·03 seconds in the human heart and is accounted for by the delay in transmission from aorta to carotid. The dicrotic incisure is also delayed and by approximately the same time interval.

The radial pulse curve.—The rise of radial pressure occurs approximately 0·10 seconds after the rise of carotid pressure.

Intra-auricular pressure curve.

Amongst the earliest studies of intra-auricular pressure are those of Chauveau and Marey (*48*, *49*, *536*, *538*); later came the observations of Fredericq (*185*, *189*), of Porter (*608*), and others (*179*, *194*). In regard to the chief waves of pressure these writers are in general agreement, but the apparatus which they used was relatively insensitive. Much more accurate curves have been obtained of recent years by Straub (*711*), Piper (*600*, *601*), Wiggers and others (*105*, *213*, *772*).

The pressure changes in the two auricles are similar. The curve consists of three chief upstrokes and three chief downstrokes, the three positive and three negative waves described by Porter and found by almost all workers.

The *first positive wave* corresponds in time to the contraction of the auricle and is universally attributed to this event, for it disappears from the curve when the auricle is thrown out of action by tetanisation and persists if the ventricle ceases to beat (*185*).

The *second positive wave* coincides precisely with the onset of ventricular systole. It is due to ventricular systole and disappears if the ventricular contraction fails. It is due to shock transmitted from the ventricle at the moment of closure and tension of the auriculo-ventricular valves, and has been ascribed to upward ballooning of these valves.

The *third positive wave begins* during the progress of the ventricular pressure plateau, it is a stasis wave and due to the accumulation of blood which enters and is held by the auricle during the ventricular systole. The rise of pressure is slow and in many records a notch appears upon it; this notch falls at the time of the second heart sound when the ventricle enters its diastole. A factor which possibly contributes to the later part of the wave is the sudden release of the base of ventricle at its diastole. The wave ends sharply at the opening of the *A-V* valves.

The *first and second negative waves.* These waves form in many curves a continuous downstroke which is broken by the sharp second positive wave. The downstroke as a whole is ascribed to three factors, namely, relaxation of the auricular wall, the enhancement of the negative pressure in the chest consequent upon ventricular systole, and the drag exerted upon the

auriculo-ventricular ring by the ventricle as it enters systole. Of these causes the first is most active in the early stages of the fall, and the last two in the later stages.

The *third negative wave.* With the diastole of the ventricle, the pressure within its cavity falls rapidly, while pressure in the auricle is still mounting. The time comes when the two pressures become equal and an instant later the *A-V* valves open and the blood pours from auricle into ventricle, while the intra-auricular pressure is relieved. The depression of the intra-auricular pressure curve which begins at the opening of the *A-V* valves is universally attributed to this cause.

The venous curve.

The pressure changes in the great veins near the auricles are very similar to those seen in intra-auricular curves (*101*, *772*). The first and third positive waves are of similar origin in the two curves; but their appearance is a little delayed. The second positive wave is modified; it is probably supplemented by a shock transmitted from neighbouring arteries. Thus Wiggers (*772*) finds two waves, the first corresponding to the intra-auricular wave and the second which he attributes to arterial shock.

The venous curve as it is recorded clinically is in the main a volume effect, and these changes in volume are largely, though probably not wholly, dependent upon the pressure changes in the right auricle. The usual clinical curves are crude, though we now possess a number of records taken with more sensitive apparatus.

The venous pulse usually comprises three chief elevations and three chief depressions. They are spoken of in Mackenzie's terminology as the waves *a*, *c*, and *v*, and the depressions *x*, *x'* and *y* (see Fig. 10). These waves and depressions do not fall at the same time instants as the elevations and depressions of intra-auricular pressure; they occur later.

The *a* wave is the result of auricular systole. The veins swell because the entry of blood to the auricles is opposed by auricular contraction; it may be also that the wave is in part produced by some reflux of blood from the auricle to veins. Taken at the root of the neck it lags behind the corresponding wave on the intra-auricular pressure curve by an interval of a tenth of a second or a little less.*

The wave *c* as it is taken in the neck has been the subject of much discussion. In many curves it is due in large part to shock transmitted from the carotid artery, and in most curves it coincides precisely with the stroke of this vessel. Exceptionally, as Bard first pointed out, the venous wave precedes the carotid upstroke by a small interval. In many curves taken from the neck there is, in all likelihood, a real venous representation;

* Morrow (*564*) estimates the rate of propagation in the veins at 1-3 metres per second.

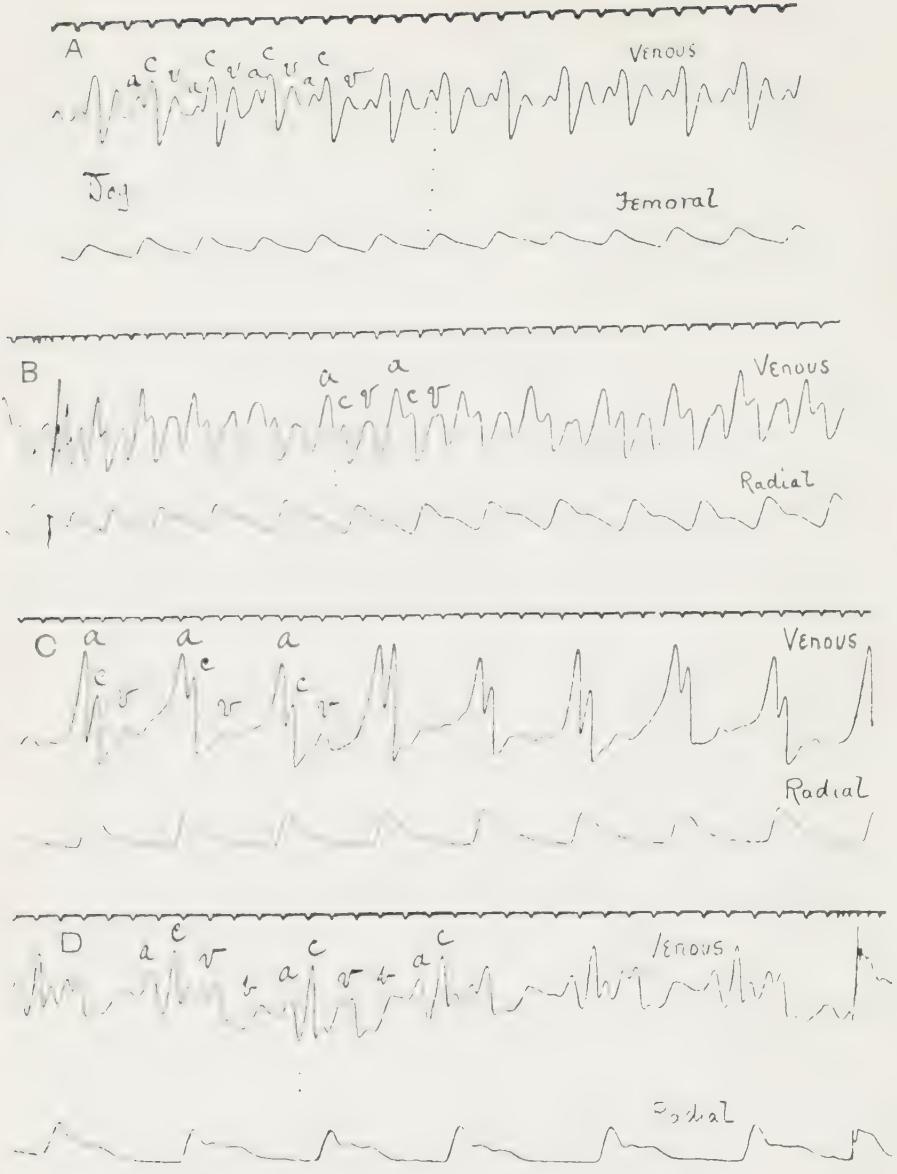


Fig. 11. Physiological forms of the venous pulse.

Polygraphic records.—*A*, simultaneous femoral and jugular curves taken from a dog. *B*-*D*, simultaneous radial and jugular curves from human subjects; *a*, *c*, and *v* waves are clearly shown in each curve. In *D*, *v* is split, and several additional waves, including *b*, are present. Note the constancy of form from cycle to cycle in each instance. The time in this and subsequent polygraphic figures is marked in fifths of a second.

but in most curves the *c* wave is probably composite, including both venous and arterial elements; in some curves, especially those which are taken with the receiver pressed more firmly on the tissues the wave is chiefly arterial. Especially in delicate curves two waves may be seen, the first of venous and the second of arterial origin (*632, 772*).

The wave *v* in the veins is normally a pure affair of stasis, the blood accumulating in the veins while the ventricle is in systole. It is said that the delay in the appearance of *v* is somewhat greater than that of *a* (*801*). In many crude clinical curves it is double.

For clinical purposes the onset of *a* is the chief event in the venous curve, for it indicates contraction of the auricle. The *a-c* interval, the time distance between the upstrokes of the corresponding waves, is used to indicate the auriculo-ventricular systolic (*As-Vs*) interval, which in its turn is taken as a measure of the capacity of the tissues to conduct impulses from auricle to ventricle. The *a-c* interval, which normally varies between 0·1 and 0·2 seconds, is of great clinical value. Its measure is, of course, relative rather than absolute; it gives no exact measure of the *As-Vs* interval, but if the *As-Vs* interval varies materially from case to case or in one case, corresponding differences will be found in the *a-c* interval.

In clinical venous curves, especially when the ventricular rate is slow, the line of the tracing often exhibits a general rise from the end of the depression "*y*" to the commencement of the next *a* wave; this ascent of the line is clearly seen in Fig. 11, *C* and *D*. It is evidently due to stasis and was termed by Morrow the second onflow wave;* the venous volume increases as the ventricle fills and the pressure in its cavity rises. Often the greater part of the ascent occurs in early diastole and the volume is then maintained; in these circumstances a distinct shoulder occurs on the diastolic portion of the curve (see Fig. 11*D*). In early diastole too, a secondary wave known as *b* or *h*, first described by Gibson (*225*) and Hirschfelder (*314*), is often seen when diastole is long.* It is said to result from a floating up of the tricuspid segments and consequent closure of the valve in early diastole as the ventricle fills with blood. According to Eyster (*156, 157*) it occurs synchronously with or a little after the third heart sound,† and this sound is also attributed to secondary closure of the auriculo-ventricular valves.

* Similar waves are found in intra-auricular pressure curves.

† For an account and records of which see *113, 225, 458, 722*.

CHAPTER III.

GALVANOMETRIC METHOD.

THE history of electrocardiographic studies will be found in the original papers referred to in this and the succeeding chapter. The main steps may be described quite briefly. In 1856, Kölliker and Müller (*389*) first demonstrated the presence of a current of action in the heart; and they were able, by laying a frog nerve-muscle preparation in contact with a beating heart, to show the presence of two distinct electrical discharges at each beat of the ventricle. Their observations were followed by those of a number of investigators (*126, 685, 686,*) notably Burdon Sanderson, working with the earlier types of rheotome and galvanometer. At a later period the capillary electrometer was used, and, employing this instrument, A. D. Waller (*745*), in 1887, first showed that it is possible to register the human heart beat. The first satisfactory curves from the mammalian heart were obtained by Bayliss and Starling (*27*).*

In 1903, Einthoven (*110, 115*) introduced his new instrument, the string galvanometer. The facility with which this instrument may be employed, and the precision of the curves obtained with it, is such that it has rapidly superseded other forms of sensitive galvanometer. It is an instrument which may be used as a routine method of recording the heart beats in experimental researches, and its introduction has brought the systematic electrical examination of patients within the field of practical medicine.

The string galvanometer.

Galvanometric instruments are based upon the principle of the interaction of a magnet and a conductor of current. In the familiar Kelvin galvanometer of the physiological laboratory, a small magnet to which a mirror is attached is suspended by a fine thread. The magnet is surrounded by coils of wire, and with the passage of currents through the latter the magnet is deflected, and a beam of light reflected from the mirror serves as an index of such movement.

The string galvanometer, in its present form the invention of Einthoven, is built on the opposite principle. A straight conducting

* In studying the earlier work on the electro-physiology of the heart, the following additional papers (*122, 534, 535, 542, 746, 748*) should be consulted.

strand lies between the two poles of a powerful magnet. Currents passed through the string induce deflections of it. The sensitivity of the instrument and the quickness of the movements have been increased by decreasing the weight of the string and by augmenting the strength of the magnetic field in which it lies. The poles of the magnet are closely approximated, so that

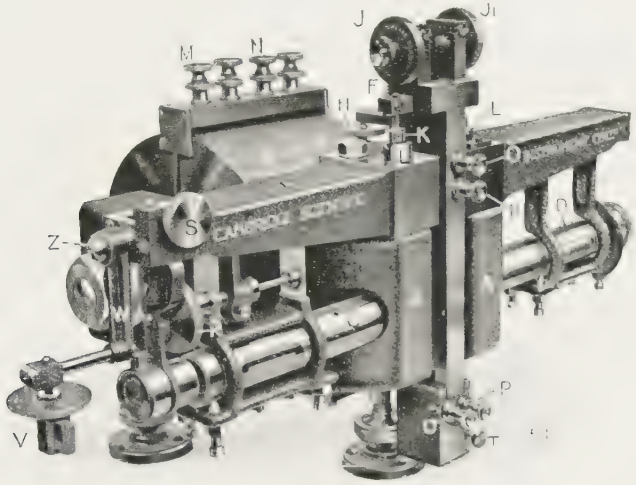


Fig. 12. The latest model of the Einthoven galvanometer. *A A* are the poles of the magnet; *B B* are the coils of the magnet, *M N* the terminals conveying constant current to it. *D* receives and condenses the beam of light which falls upon the string or strings through a mica window in the carrier. *C* carries the projecting lens, and the same tube has, at its exposed end, an eye-piece which focusses the light on the camera; *R* is a focussing screw; *S* moves the microscope from side to side; *W* is a stand carrying two prisms at *V* and is used to converge the images of two strings when these are employed. The carrier has a number of adjusting screws; *H* swings the whole carrier on pivots (*K* and *L*) moving the mica window to and fro across the beam of light; *J* and *J′* alter the tensions of the strings; *T* moves one string and thus adjusts the relative position of the two strings; *O O* and *P P* are the terminals connecting to the ends of the wires and through them the currents to be tested are conveyed. Carriers may be used which are fitted with a single string or with two strings.

only a narrow chink separates them, and the field is saturated. It is in this cleft that the string is fixed, and its movements are observed by projecting its shadow upon a screen. The strings employed are extremely delicate, consisting of finely drawn platinum or of a film of silver over a finely drawn quartz or glass thread. In thickness the fibre is 0·002 to 0·005 mm..

The Cambridge model of this instrument* is shown in detail in Fig. 12; in this model two strings are held in a carrier (*J to O*) pivoted above and

* The galvanometric outfit here described has been specially adapted for clinical studies and experimental work by the Cambridge Scientific Instrument Company acting in co-operation with my laboratory.

suspended between the poles of the magnet (*A A*). The carrier encases the strings and protects these delicate fibres from air currents and dust; it has attached to it the terminals which convey the currents to be tested (*O-O* and *P-P*) and milled screws (*J* and J^1) for adjusting the tension of the strings and their positions in the magnetic field (*H*). The circuits in practice vary in detail in different institutions and according to the purposes for which they are employed. In Fig. 13 is shown diagrammatically the Cambridge switchboard, which has been specially arranged for rapid and accurate clinical observations. The magnet of the galvanometer is shown, and the string is represented by a fine dotted line passing vertically between its poles; the ends of the string connect to the wires of the chief circuit (heavy lines). The continuity of the two main wires is interrupted, on the

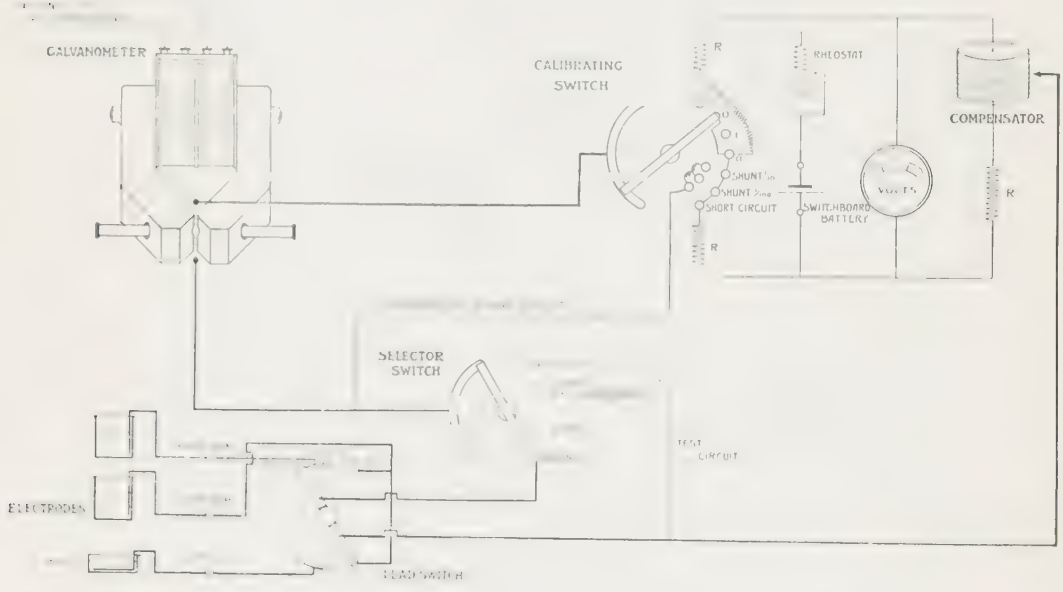


Fig. 13. Diagram of the connections of the galvanometer to the three bath electrodes for the limbs by means of the Cambridge switchboard. The arrangement of the patient is shown in Fig. 21.

one hand by the calibrating switch and compensator, on the other by the selector switch; the main wires eventually terminate in the lead switch. The purpose of the *lead switch* is rapidly to connect the two main wires to any pair of the three electrodes in which the right arm, left arm and left leg of the subject are immersed. In the position shown in the diagram, the right and left arm electrodes are in circuit (*RA-LA*). The patient's body and his two selected limbs complete a circuit with the string of the galvanometer, a circuit which, when the instrument is recording (position

figured), is complicated only by the presence of the compensator. The selector switch has four positions, (a) the recording position as depicted, (b) an off position in which the galvanometric circuit is broken ; (c) and (d) positions in which the galvanometer lies in *test circuits*, the one containing a 4000 ohm resistance, the other containing no resistance ; these circuits are used when by a preliminary test an estimate of the instrument's sensitivity to standard currents thrown into it is desired, or when an estimate of the internal resistance (chiefly string resistance) is desired. The *calibrating* switch is used for a number of purposes. When the switchboard is first connected to a patient, this switch stands on the *short circuit* stop ; thereby the delicate fibre is safeguarded against the receipt of any current from patient to compensator (any such current then flows through the short circuit wire). When all is ready, the switch is moved to the 1/100 shunt ; in this position approximately 1/100 of the current to be tested is thrown into the galvanometer (the rest passing through the shunt circuit) ; if this current is sufficient to deflect the string from its zero point, it is brought back to the zero by means of the compensator. The switch is now moved to the 1/10th shunt (which allows approximately 1/10th of the tested current to pass through the galvanometer), and any permanent deflection of the string is again compensated. The process of compensation is repeated after the switch has been moved to the first zero position, at which point all the tested heart current passes into the galvanometer. The object of this procedure will be clear when it is realised that currents of two orders are obtained from the patient : first, there is a constant current which comes from the skin and is due to activity of the sweat glands ; this constant current, if it is not compensated by stages, may be of sufficient magnitude to fracture the recording fibre ; it is neutralised and the string is maintained at zero by throwing a current into circuit in the opposite direction from the compensator ; secondly, there are the minute fluctuating currents produced by the heart beat, and these it is desired to record with the string at its most sensitive point in the magnetic field, namely, when it lies at zero. The three final positions of the switch 1, 0 and 3 are used to calibrate the excursion of the recording fibre. Movement of the switch from 0 to 1, or from 0 to 3, changes the E.M.F. on the string terminals by one or three millivolts, respectively.

Obtaining standardised electrocardiograms.

Electrocardiograms, whether clinical or experimental, should be standardised ; the standard now universally adopted is Einthoven's standard (*111, 114*), in which one centimetre of excursion in the final photograph is equivalent to one millivolt potential difference at the ends of the recording fibre ; the measurement of standard movement in terms of E.M.F. has the great advantage of neglecting the resistance of patient and the resistance of string, both of which are variable factors. The object of standardisation

is to keep records from all laboratories to the same scale of excursion, and to permit a comparison of the amplitude of the corresponding deflections, in a given patient from time to time and in different patients. It is effected most simply and with sufficient accuracy for clinical purposes, by introducing the patient into circuit, compensating the skin current, and, lastly, by increasing the sensitivity of the fibre (by slackening it) until the introduction of three millivolts deflects the moving string through three centimetres (see Fig. 14).

Testing the properties of the string.—If standard curves of correct form are to be obtained, the response of the instrument to simple currents must be tested from time to time (*463*, *680*); for the excursion and shape of the electrocardiogram may be modified by the properties of the string and the conditions of the observation.

If an E.M.F. of one millivolt is thrown into circuit (*478*, *680*) while the patient is disconnected and the tension of the fibre is so arranged as to give an excursion of one centimetre, the curve obtained should be similar in outline to those shown on the right hand of Fig. 15. The string moves when the current enters it and takes up a new position one centimetre away. In arriving at the new position it describes a curve. Now the characters of this curve are important. Fig. 15 shows six electrocardiograms from the same man, and the six corresponding responses to an E.M.F. of one millivolt. The curves differ because they were taken with different resistances in circuit and consequently with different string tensions.* From above downwards, the resistances were increased and the string slackened correspondingly, all the curves being taken so that the standard deflection of one centimetre was obtained when one millivolt was introduced (deflections to right of strips).

In the first place, the movement of the string in response to one millivolt should be "dead beat"; it should not overshoot its new position. Overshoot occurs when the string is too tense (as in Fig. 14, at *b*), or when the recording fibre is too heavy. The defect of overshoot is not observed in the Cambridge instrument, in the conditions under which it is used for clinical purposes; but in many instruments on the market it forms a prominent defect, increasing the excursion of the sharp deflections.

In the second place, the movement should be of sufficient rapidity, the slacker the string, the more slowly does it respond; the deflection times for the six strips of Fig. 15 are 0·013, 0·023, 0·028, 0·045, 0·060 and 0·075 seconds, respectively, from *A* to *F*. Now the initial changes in the currents to be recorded from the heart are rapid and, if the quickest movement of which the string is capable is too slow to follow these changes, accuracy is lost. The distortion of the curves, especially of their rapid initial phases as the string is slackened, is well illustrated in the accompanying figure. The

* Adding resistance to the main circuit decreases the sensitiveness of the recording instrument and the string must be slackened to compensate this decrease so that the previous excursion may be obtained again.

Fig. 14. Portions of a continuous curve illustrating the process of standardisation. This and all curves of the book read from left to right. The string is at first still at the zero position: at *a*, a shunt is removed and the string is deflected by the skin current; at the same time, small deflections due to the heart beat are recorded. This deflection is compensated by throwing in current from the compensator (at *b*). A further shunt is removed at *c*, the skin current requiring in this instance no further compensation; at the removal of the shunt the electrocardiogram grows in height. The string is now slackened until the magnified shadow of the base line is seen to move on the scale of the camera, through exactly three centimetres when three millivolts are thrown into circuit.

upstrokes and downstrokes are rendered more oblique and their amplitudes are decreased. The first two curves are identical because the degree of slackness reached by the string is as yet insufficient to cause distortion.

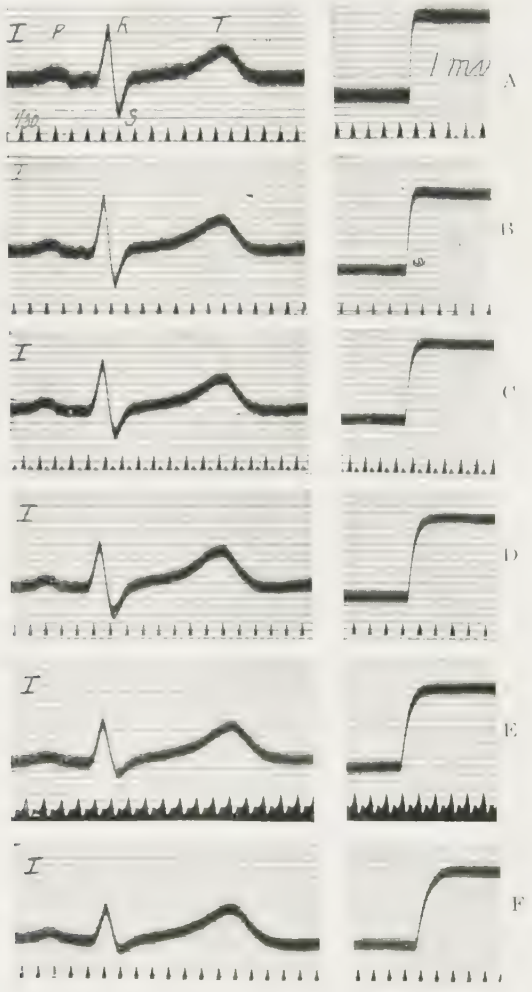


Fig. 15. Six electrocardiograms from lead *I* and from a single subject, and the six corresponding deflection curves, to illustrate the distortion of curves when the string tension is too slack. As the string is slackened beyond a certain limit and the deflection time (the time of response to an *E.M.F.* of one millivolt over the excursion of one centimetre) increases. *R* and *S* are materially reduced in amplitude. Time in thirtieths of a second. In this and subsequent figures the lead from which the curve was taken is indicated in the left top corner.

There is an upper limit of tension and a lower limit of tension in sound instruments, the former yielding overshooting, the latter insufficient speed of movement; between these limits accurate observations are alone obtained. For clinical purposes the deflection time should be at least as low as 0·02 seconds (*478*), when a resistance of 4,000-10,000 ohms is added by means of the test circuit. This condition is more than fulfilled by the instrument here described of which the natural frequence is approximately 200-250 per second.

Suitable contact electrodes.—The electrodes advocated for making contact with the patient are shown in Fig. 21. A porous inner vessel is filled with warm water, salt and well-washed cotton wool,* to give a mixture of porridge-like consistence; this is surrounded by an outer vessel containing saturated zinc sulphate in which a sheet of zinc is immersed to which the leading-off wire is soldered. Each electrode should be insulated from the floor. These electrodes are non-polarisable. That it is necessary to employ non-polarisable as opposed to polarisable electrodes in accurate electrocardiographic work may be shown readily (*473*, *681*).

1. If an E.M.F. of one millivolt is introduced (Fig. 16, curve 1, *in*) into the simple closed circuit of a properly tuned string galvanometric recorder, and is cut out later (*out*), the record is as depicted. It represents, as accurately as modern instruments will permit, the flow of current through the fibre. If the millivolt is introduced into the same circuit, in which is interposed a pair of non-polarising electrodes, separated by a fixed interval of normal saline, then, providing string tension and total circuit resistance remain unaltered, *an identical record is obtained* (curve 2). But if, in similar circumstances, *polarisable* electrodes (of platinum) are utilised, *the record suffers distortion.* If the electrodes polarise slowly, distortion is present (curve 3); if they polarise rapidly, distortion is the greater (curve 4). Distortion is due to the rapid development and maintenance of a charge on the surface of the electrodes, while current is flowing through them. In the second figured instance of polarisation (curve 4), (*a*) the amplitude of the initial deflection, produced by the E.M.F. introduced, is materially reduced, owing to the rapidity with which polarity develops; (*b*) although the E.M.F. is maintained, the string returns to zero, and (*c*) on cutting out the E.M.F., the string is deflected across the zero line, by the now unbalanced charge on the electrodes. All these effects are clearly *defects* in the record.

If with readily polarisable electrodes a true record of a simple current change is unobtainable, it is unreasonable to believe that complex currents developed by the heart beat will be accurately recorded. In point of fact,

* Warm water adds to the patient's comfort and decreases muscular tremor; salt decreases resistance; cotton wool supports the limb and decreases movement of the limb and of the surface of the fluid.

the resultant distortions can be predicted. The upstroke of an initial deflection R will be reduced in amplitude because it will be neutralised by the developing polarity. As R subsides, the string will deflect across the zero line and will produce an artefact resembling S (if S is not present) or exaggerate S (if S is present); the final summit T will be similarly reduced in magnitude by neutralisation, and will be followed by an artificial depression as T subsides. That such distortion actually results is clearly shown by curves 5 and 6 in which each of these changes is to be observed.

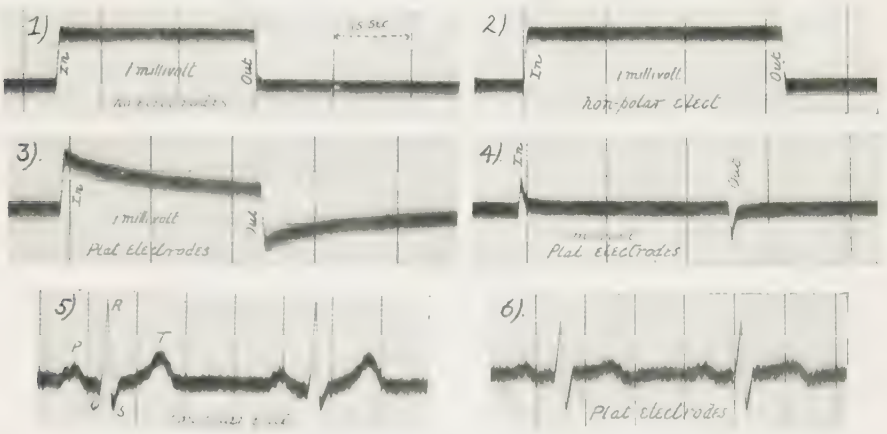


Fig. 16. (*Journ. of Physiol., 1915, XLIX. Proc. physiol. soc., L.*). 1. One millivolt was introduced into, and cut out of, a simple closed circuit. Circuit resistance = 4800 ohms (string 2800 ohms; added resistance 2000 ohms). The excursion of the fibre is "dead beat" as it should be, and 10 scale divisions in amplitude. 2. One millivolt similarly introduced into, and cut out of, the same circuit containing non-polarising electrodes. Total circuit resistance 4800 ohms (string resistance 2800 ohms; electrodes and salt solution 1850 ohms; added resistance 150 ohms). 3 and 4. One millivolt introduced into, and cut out of, the same circuit containing polarising platinum electrodes (larger and smaller electrodes); total circuit resistance 4800 ohms (string 2800 ohms; electrodes and salt solution 250 ohms; added resistance 1750 ohms). The sensitivityof the string was constant throughout the series. 5. Human electrocardiogram taken by means of the non-polarisable electrodes (of 2); standardisation so that 10 scale divisions = 1 millivolt. 6. Curves from the same heart and lead, taken with polarising platinum electrodes (of 4). The total circuit resistance and string sensitivity were identical in the case of the two electrocardiograms. Time in fifths of a second.

In electrocardiography the use of electrodes which polarise appreciably is indefensible, because it is impossible to accept the standardisation of the corresponding curves against a known E.M.F., and because the curves themselves are apt to suffer distortion.*

* As Pardee (*586*) has recently, and quite rightly, said, the polarisation effect is greater when the electrode surface is small (the electrodes in Fig. 16, *3*, were larger than in Fig. 16, *4*). If large simple metal contacts such as he advocates are used, it is quite true that no material polarisation is usually obtained; but from time to time, and for reasons not fully determined, the polarisation of such electrodes is considerable and definitely affects the values of the curves.

To test the electrodes it is sufficient to connect the two inner porous pots by means of a strip of washed cotton wool soaked in brine and, placing this pair of electrodes in the main circuit, to introduce an E.M.F. of one millivolt; the resultant deflection should be permanent, the string should show no tendency to overshoot nor to return towards the zero line.*

Photographing.—The string lies vertically and its shadow is projected by an optical system (Fig. 17), consisting of arc light, condenser and

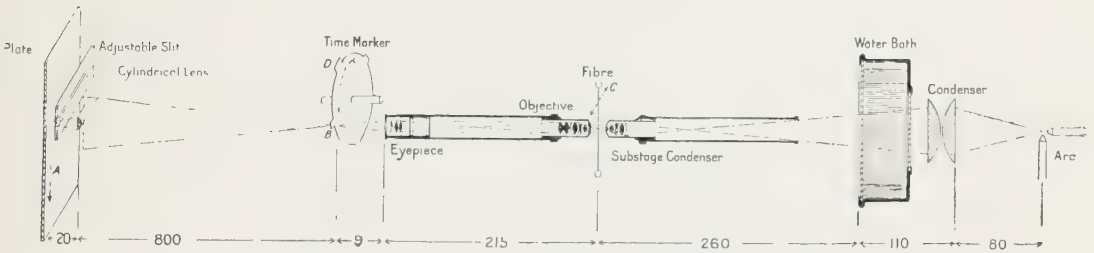


Fig. 17. Sectional view of the optical arrangement, utilised in taking galvanometric records. The light derived from an arc passes through a first condenser and a cooling bath and is focussed upon the fibre by means of a substage condenser. The image is projected by the objective and eye-piece on to the cylindrical lens; *en route*, the beam of light is cut at *B* (where it comes to a focus) by the teeth of a rotating disc, the time-marker. The direction of the fibre movement is across the beam of light as indicated by the arrow *C*; the shadow of the fibre is projected upon the cylindrical lens; it is vertical, cutting the cylindrical lens at right angles; its movement is in line with the axis of the cylindrical lens and the adjustable slit. The plate which records these movements runs vertically from above downwards (arrow *A*).

microscope. The magnification adopted is usually 600 diameters. The vertical shadow falls upon a cylindrical lens, whose axis is horizontal and which focusses the light, cut by this shadow, upon a photographic plate; the latter moves inside the camera, its movement being controlled by an oil cylinder (Fig. 18), and moves vertically behind a slit in which the cylindrical lens is fixed. The lens is ruled at millimetre distances, and these lines are photographed on the plate as it travels; these are the horizontal lines on the illustrations as they are published.† The shadow of the string moves from side to side, leaving upon the developed plate a white line, which varies in thickness according to the rate at which the string is moving. This line is black and its movements are up and down in the reproductions. The speed of the plate is under control, as is also the amount of light admitted

* According to Pardee (*587*), such overshooting may sometimes take place when non-polarising electrodes are used. It is seen when skin resistance is high, and, according to Pardee, is possibly attributable to the skin acting as a condenser surface.

† Certain of these figures have been reduced in size for publication; the values of the deflection are to be measured therefore against the millimetre lines and not by direct measurement.

to the camera. For clinical purposes a shutter is employed by means of which three records may be successively taken side by side upon a single plate.*

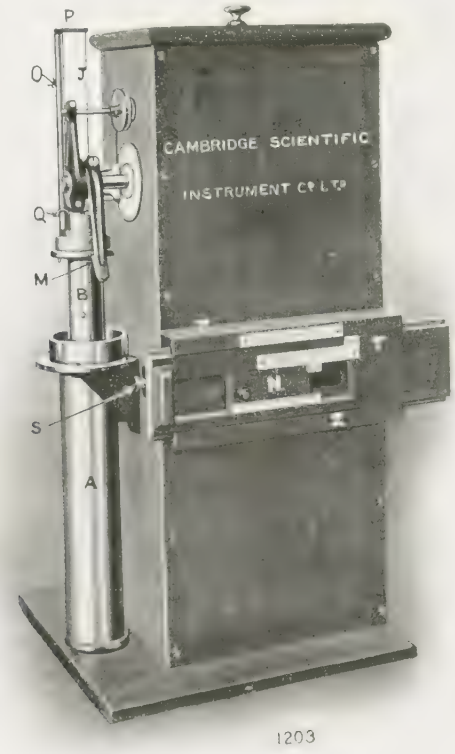


Fig. 18. The plate camera of the Cambridge outfit. *A* is a cylinder containing oil, *B* a hollow plunger which as it descends takes up the oil; the rate at which the descent is accomplished is altered by a screw vernier at *Q* which opens or closes the communication between *A* and *B*. *M* is a starting release. The plate moves in a carrier inside the camera, its movement being controlled by the oil piston through pulleys (*P*, *J*); it is exposed behind the shutter. The latter consists of a slide, *T*, running in the grooves of *Z*, *Z*, and a second slide, *N*, with scales attached. The amount of plate exposed is controlled by the slide *N*, the portion of plate by the slide *T*. As figured, one-third of the plate would be exposed, the gap through which the light enters the cylindrical lens being seen in the illustration.

Time-markers. — The time-markings in the records of this book are various, because the curves have been taken during the development of the apparatus. The modern time-marker is a rotatory marker. A toothed

* With good lighting I have found "Process" plates to give the best results. They are very slow, and consequently require excellent illumination; they give great contrast and ensure black and white figures.

wheel, driven by an electric current, and controlled by a tuning fork, revolves and cuts the beam of light where it is brought to a focus after it leaves the eye-piece of the microscope. As each tooth passes the eye-piece, it cuts off all light from the cylindrical lens for an instant, ruling a clean line across the plate (this line is vertical and black in the reproductions : see Fig. 26).

Simultaneous records.—Many methods have been devised for securing simultaneous electrocardiograms. Bull (*44*) used two galvanometers arranged in tandem fashion, the images of the two strings being projected upon the camera by the same eye-piece. The optical adjustments in this scheme need to be very exact.

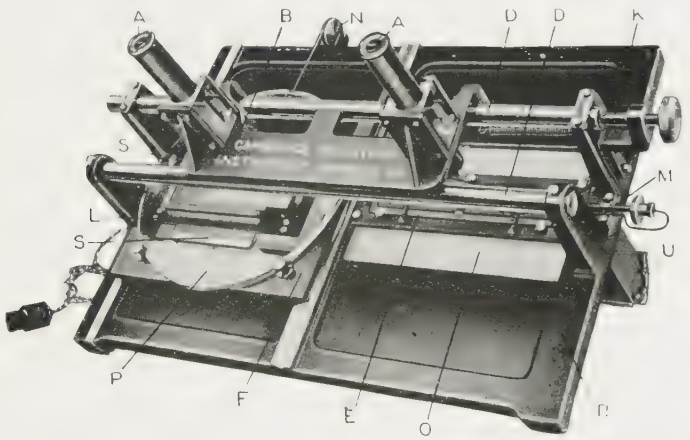


Fig. 19. The Lucas co-ordinate comparator. Upon a rigid cast-iron stand, *R*, two steel bars (*D D*) are fixed, and upon these a carrier, *B*, which supports the microscopes (*A A*) slides from side to side. Fine lateral adjustments of the carrier are obtained with the screw, *K*, Through the right-hand microscope a finely divided millimetre glass scale is read ; the scale is fixed in a frame, the lower edge of which is seen at *E* ; this scale may be adjusted laterally by means of the screw, *M*. The photographic record is placed on a circular glass frame, *P*, which may be rotated so that the lines of the plate are exactly horizontal. The glass frame rests on a metal carrier, *F*, which moves up and down against a counterpoised weight over the pulley, *N*. The background is illuminated by electrical lamps held in the shield, *U*.

Two galvanometers have been used, side by side, each with its own projection system (*461*), the light from the two being kept apart up to the point where it falls upon the cylindrical lens. The disadvantage of this system is that the rotatory time-marker cannot be employed.

Recently the Cambridge Scientific Company has introduced a single carrier fitted with two strings, the images of which are eventually brought conveniently near to each other by an arrangement of prismatic lenses

(see Fig. 12). It is perhaps the most practical device of its kind, and by means of it the simultaneous records shown in this book have been taken for the most part.

By placing a moving lever immediately in front of and vertically at right angles to the cylindrical lens, additional records, of arterial pulse, venous pulse, respiration, blood pressure, etc., may readily be obtained, simultaneously with the electrocardiogram. Parkinson (*588*) has recently projected arterial and venous pulsations directly by placing the patient between the eye-piece of the microscope and the camera. The shadow of the skin, where it pulsates, is thrown directly upon the cylindrical lens and the movement is magnified in the record by this optical projection. Such records are both accurate and beautiful.

Measurement.—Minute measurements are made from the negatives by means of a comparator (Fig. 19). This instrument consists essentially of two microscopes of small magnifying power and fixed in a rigid carrier which slides horizontally on two steel bars. They are moved laterally by means of a millhead screw. Under one microscope is an adjustable plate holder, under the other a divided millimetre scale. Measurements in millimetres are taken of the intervals between the vertical lines representing time, and between the desired points on the electrocardiogram; the last named are then converted to seconds. By means of this device, readings from good curves may be obtained with an error of 1/1000 of a second or less.

Registration of heart sounds.—The method which I have employed (*461*) in registering heart sounds is very similar to that described by Einthoven (*112*) and Fahr (*166*). A large and sensitive microphone (*M* in Fig. 20) communicates with the air through thick-walled rubber tubing and a stethoscope end-piece (*S*). The tubing has a second outlet (*O*), which remains permanently open; its purpose is to cut out the changes of pressure in the system of tubing which would otherwise result when the stethoscope is applied over an area of chest wall which is pulsating. The microphone circuit includes a dry cell, a make-break key (*K2*), a rheostat ($R = 70$ ohms), and the primary coil (10 ohms) of an inductorium or transformer (*T*). The secondary coil (coreless, 5,300 ohms) of the transformer is connected to a short-circuiting key (*K1*); this key, when closed, forms a shunt to the fibre of the string galvanometer (*G*). Sound vibrations transmitted through the stethoscope fall upon the microphonic plate, and by pressure produce variations in the internal resistance of the microphonic circuit; the current flowing through the circuit varies accordingly and, varying, induces currents of higher potential in the secondary circuit; these secondary currents flow directly through the string when the shunt (*K1*) is open. A tense string is employed, and sound vibrations having a frequence of from 200-300 per minute are recorded without material damping.

The double fibre carrier is employed, one string being used to record sound, the other to record the electrocardiogram.* Fig. 27 is an example of a simultaneous electrocardiogram and the sounds recorded from the apex beat of a normal subject.†

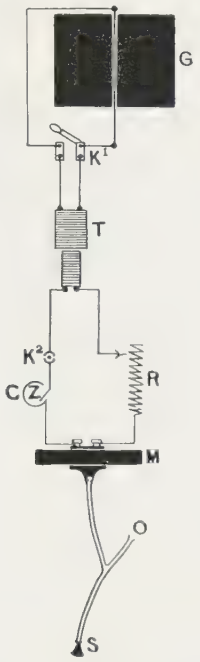


Fig. 20. (*Quart. Journ. Med., 1912-13, VI, 441, Fig. 1.*) A diagram illustrating the apparatus used in taking heart sounds records.

* In the earlier observations of which Fig. 27 is an illustration, I used separate galvanometers.

† Other papers on heart sound records will be found in references *120*, *223*, *364*, *365*, *751* and *752*.

CHAPTER IV.

THE BROAD FEATURES AND TIME-RELATIONS OF THE NORMAL ELECTROCARDIOGRAM AND CERTAIN PRINCIPLES OF INTERPRETATION.

The broad features of the normal electrocardiogram.

The leads adopted.— Electrocardiographic curves may be obtained by leading from various points of the body. In examining clinical subjects, Einthoven (*111*) adopts three leads, taking in pairs, the right arm—left arm (lead *I*), the right arm— left leg (lead *II*), and finally the left arm—left leg (lead *III*); these leads have come into general use.

It should be understood that the type of electric curve obtained from the heart depends largely upon the lead chosen, and that no two leads yield exactly the same picture (Fig. 21). The reason for this change with the leads will be explained at a later stage; we may be content for the moment in noting it. Each lead is serviceable in given circumstances, for one lead may give information which another will not. All indicate the systole of both the auricle and the ventricle.

The physiological type of human electrocardiogram.—The electrocardiogram of man, like that of other mammalia, consists of two parts, an *auricular complex* and a *ventricular complex*. As a whole the electrocardiogram exhibits considerable variations in form from subject to subject even in health, but in a single subject it is constant under given conditions; thus, electrocardiograms might be successfully adopted as a means of identification (*478*). In this chapter we shall consider briefly certain variations in the form of the physiological electrocardiogram.

The *auricular complex* consists of a primary deflection in the upward direction. Adopting the terminology of Einthoven, it is termed the summit or peak *P* (Fig. 29). This summit, which is either rounded or pointed, is succeeded by a horizontal line (an isoelectric* period) or by a less prominent deflection in the opposite or downward direction.

* The contacts are isoelectric when no current is passing through the string.

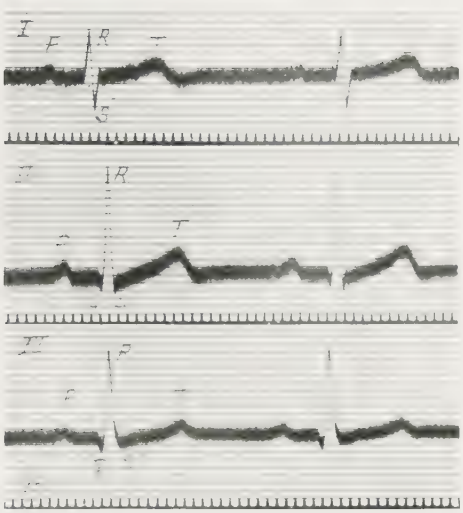


Fig. 21. Three electrocardiograms from a young and healthy subject.

I. Leading from the right arm to the left arm.

II. Leading from the right arm to the left leg.

III. Leading from the left arm to the left leg.

The three curves were taken separately and each *standardised* so that one centimetre of excursion represents one millivolt. The horizontal lines are ruled photographically at distances of one millimetre in the original curves and serve as a convenient scale of measurement; each scale division is equivalent to 1/10 millivolt. This standard has been used for all the curves of this book except where it is definitely stated to the contrary The time records thirtieths of seconds.

Note the varying amplitude of the several deflections in the separate leads, and the appearance of a deflection *Q* in leads *II* and *III* in this instance. *P* represents auricular, *Q*, *R*, *S* and *T* ventricular, activity.

FIG. 22. Photograph of a subject as connected for observation. The two arms and the left leg are used, and curves are taken from the three leads which are represented by the arrows drawn upon the figure. The zinc sulphate is placed in the outer vessels of the electrodes shown in this figure

The *ventricular complex* is, as a rule, triphasic or quadriphasic, and is constituted by the deflections R, S and T, or Q, R, S and T, of which R and T are directed upwards, while Q and S are directed downwards. R is usually the most conspicuous summit in the curve and its duration is brief (usually 0·03 seconds or less). It is often preceded by a small and brief

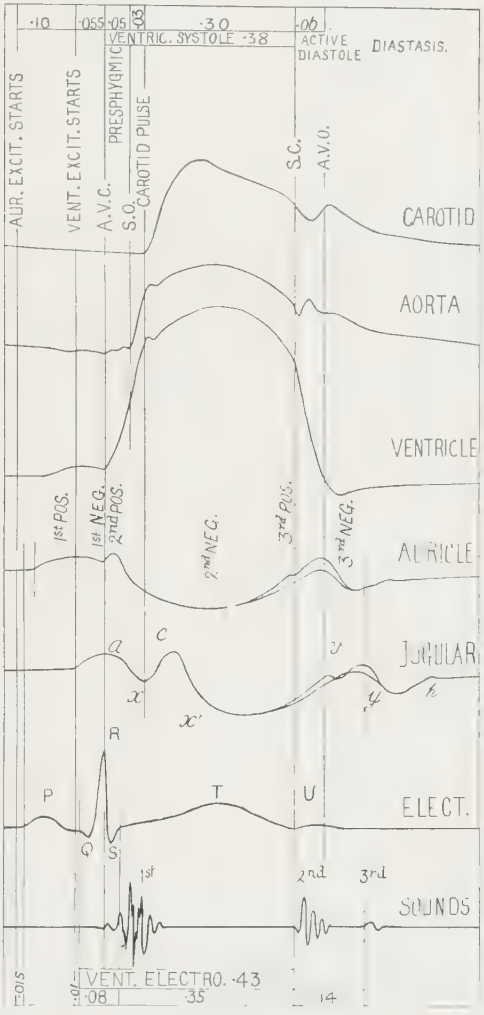


Fig. 23.

dip, Q; it is often followed by a brief dip, S, which is of very variable amplitude.

The opening phases of the electrocardiogram consist, therefore, of a summit, P, associated as we shall see with auricular systole, and summits

and dips, *Q*, *R* and *S*, associated with the initial events of ventricular systole. The *Q*, *R*, *S* group of deflections is of special importance and is one of the distinguishing features of a normal or physiological curve; as I have pointed out, *this group of deflections has a total duration of no more than 0·1 of a second, and usually constitutes less than one-third of the full ventricular complex (463).* It is followed by a larger or shorter line which is horizontal.* during which the contacts are isoelectric, and the whole complex ends in a broad and prolonged deflection, *T*.†

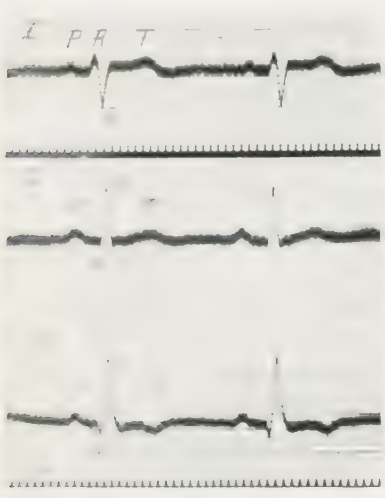


Fig. 24*a*.

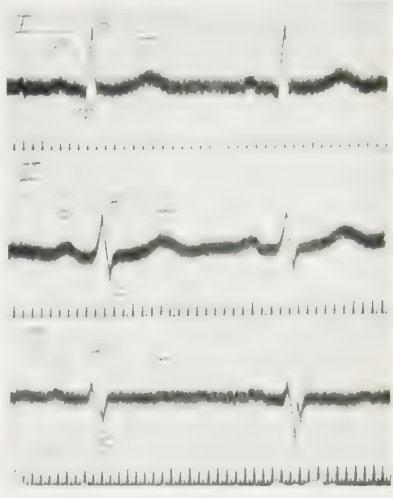


Fig. 24*b*.

Fig. 24*a*. Normal human electrocardiograms showing the tallest *R* in lead *III*, and the shortest *R* and deepest *S* in lead *I*. Time in thirtieths of a second.

Fig. 24*b*. Normal human electrocardiograms showing the tallest *R* in lead *I*, and the shortest *R* and deepest *S* in lead *III*. Time in thirtieths of a second.

Variations in the physiological type in the several leads (*477*).—As a general rule *R* is most prominent in lead *II*; but it may be largest in lead *III*, in which case it is short in lead *I*, and *S* is conspicuous in the same lead. It may also be most prominent in lead *I*, in which case it is short in lead *III*, and *S* is conspicuous in this lead (see Fig. 24*a* and 24*b*).

Normal electrocardiograms often exhibit notching of *P*, and the summit of *R*, and base of *S*, are not infrequently split in leads *II* and *III*. In lead *III* bizarre types of initial deflections (the *Q*, *R*, *S* group) are not uncommon; examples are shown in Fig. 25.

* Not infrequently absent.

† In many normal curves *T* is followed, especially in lead *II*, by a further summit *U* of small dimensions. It falls in early diastole and its meaning is not understood.

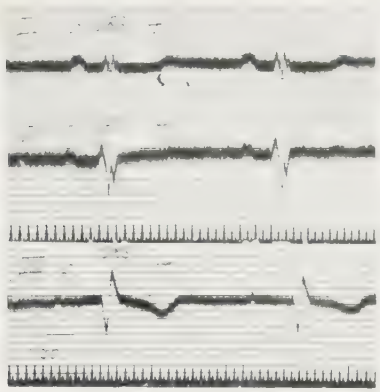


Fig. 25. Normal human curves taken from lead *III* in three different subjects. Illustrating the curious arrangements of the initial ventricular deflections which sometimes occur in this lead. They are often associated with inversion of *T*. Time in thirtieths of a second.

In the accompanying table the minimum and maximum and average values of the several deflections of the normal electrocardiogram, as they were found in 52 healthy subjects, are given (*478*).

	P	*Q*	*R*	*S*	*T*	*U*
			Lead *I.*			
Minimum	Trace	0·0	1·5	0·0	—0·5*	0·0
Average	0·52	0·51	5·16	2·06	1·93	0·10
Maximum	1·0	2·0	12·0	6·0	5·5	Trace
			Lead *II.*			
Minimum	Trace	0·0	4·0	0·0	Trace	0·0
Average	1·16	0·73	10·32	2·23	2·46	0·16
Maximum	1·7	2·5	16·5	4·5	5·0	0·8
			Lead *III.*			
Minimum	Trace	0·0	2·0	0·0	—2·0	0·0
Average	0·81	0·86	6·61	1·73	0·61	0·06
Maximum	1·5	2·5	14·0	4·0	3·0	0·3

The chief time-relations of the electrocardiogram.—Electrocardiograms have been taken simultaneously with records from the auricular and ventricular musculature (Fig. 26), with intra-ventricular pressure curves and with heart sound (Fig. 27) and polygraphic curves (Fig. 28) on numerous occasions. The records taken by different methods and by different workers are in fairly close agreement. They are portrayed in the diagram (Fig. 23).

* The — sign indicates inversion of the deflection.

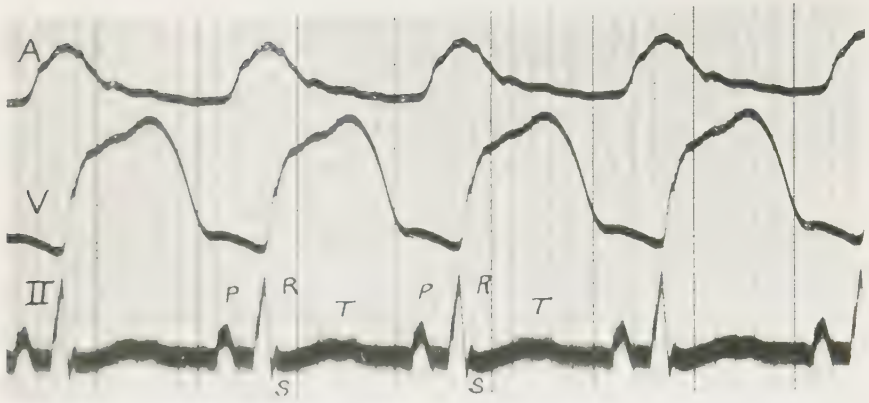


Fig. 26. Simultaneous electrocardiogram and myocardiographic curves, the latter taken by levers attached directly to the wall of the exposed heart. Taken from a dog. The vertical lines represent the time-marker, which records 1/5 and 1/25 seconds. The lines were ruled photographically while the curve was taken, and each cuts the three curves at precisely the same instant in time. The relation between *P* the auricular summit and the upstroke of the auricular myocardiogram (*A*) (upstroke representing systole) and the relation between *R* the first ventricular deflection and the upstroke of the ventricular myocardiogram (*V*) is displayed.

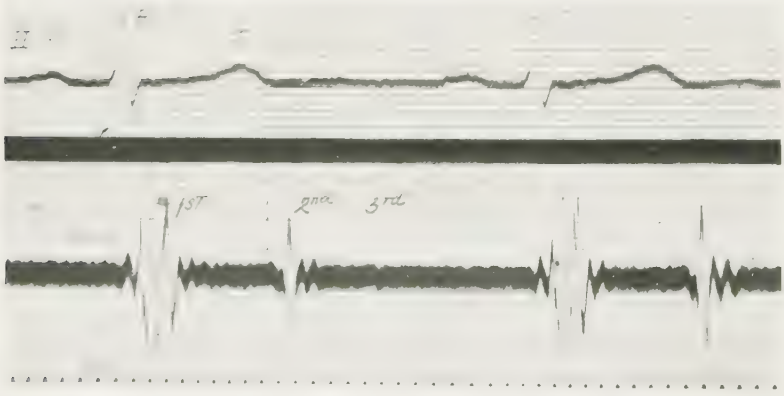


Fig. 27. Simultaneous electrocardiogram and heart sound curve from a normal human subject. The figure shows the time-relations of the electrocardiogram to the beginnings of the 1st and 2nd heart sounds. All points on a vertical line are simultaneous. Time in thirtieths of a second.

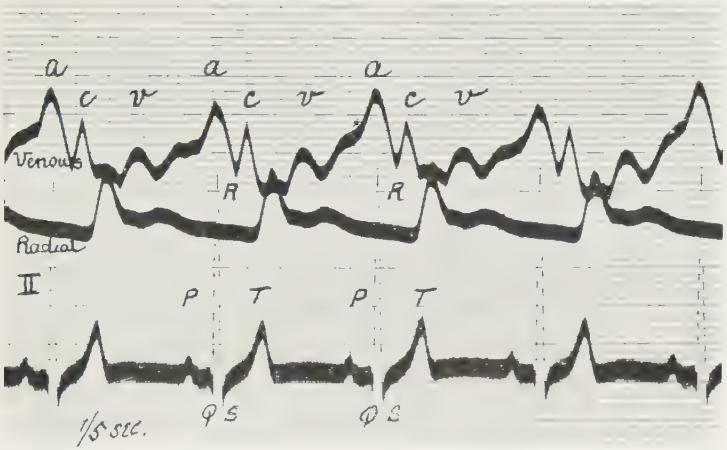


Fig. 28. Simultaneous electrocardiogram, venous and radial curves from a young man. On account of the conduction of the polygraphic curves through the air of the rubber tubing, the venous and radial records are displaced to the right equally and by approximately 0·03 of a second. This delay may be allowed for all similar curves in this book. The relations between the several waves and deflections of the curves may be gauged if this delay is allowed. Time in fifths of a second.

P stands in relation to auricular systole and its upstroke precedes *R* by from 0·13 to 0·21 of a second, this time interval constituting the *P*-*R* interval (observations upon 52 healthy young men) (*478*). Usually the interval lies between 0·13 and 0·16 of a second.* In dogs, the *P*-*R* interval is between 0·08 and 0·10, and in cats between 0·06 and 0·08 of a second. The upstroke of *P* precedes the upstroke of *a* in the human jugular curve by from 0·1 to 0·15 of a second. In six dogs the upstroke lay from ·024 to ·043 of a second before the commencement of the curve of auricular shortening.

The upstroke of *R* precedes the upstroke of *c* in the human jugular by from 0·1 to 0·15 of a second. The beginning of the initial ventricular deflection (*R* or *Q*) usually precedes the onset of ventricular contraction (as estimated from myocardiograms from the front of the ventricle in six dogs) by from 0·020 to 0·038 of a second. This interval has been regarded as a measure of the latency of the contraction. But it has been shown by Kahn (*363*), and the writer is in agreement with him, that when the muscle is artificially excited in the neighbourhood of the attached myocardiographic lever, the interval is less (according to this author it amounts to 0.002 of a second or even less). There is a delay of approximately 0·02 to 0·03 of a second between the commencement of ventricular activity and its appearance and record on the surface of the heart. Kahn has recorded simultaneously

* Schrumpf (*694*), analysing the curves of 342 subjects, gives the normal interval at from 0·05 to 0·15 of a second. An interval of more than 0·15 of a second he regards as abnormal. In placing the upper limit of normality as low as 0·15 sec. Schrumpf is certainly in error.

R of the electrocardiogram and the intra-ventricular pressure rise and finds a delay of approximately this extent, but Piper (*602*) using more exact methods has recently stated the delay to be less.

The summit *T* falls during the systole of the ventricle, while the whole mass of the muscle is shortened. In comparison with intra-ventricular pressure curves it has been found to subside a few hundredths of a second before or after the end of the plateau (*212*, *213*, *358*, *602*, *777*).

The relations of the electrocardiogram to the heart sounds are perhaps the most valuable which we possess; the heart sounds are the only accurate standards for comparison in man (*25*, *166*, *359*, *364*, *458*, *461*, *778*).

TABLE I (HUMAN).

Heart rate.	Beginning of *Q* to 1st sound.	Beginning of *R* to 1st sound.	End of *T* to 2nd sound.
77	0·039	0·026	0·0
77	0·036	0·026	− 0·015*
74	No *Q*	0·009	− 0·014
100	0·015	0·008	0·0
95	No *Q*	0·005	0·002
108	0·012	0·006	0·005
75	No *Q*	0·015	0·0
68	0·015	0·005	
81	0·011	0·002	
72	0·024	0·011	0·005
102	No *Q*	0·026	0·0
90	0·028	0·018	—0·013
78	0·013	0·008	0·019
90	0·025	0·018	0·028
75	0·030	0·025	—0·035

The error in measurement in this table is probably no greater than 0·005 of a second in any instance.

In the dog I find similar relations:

TABLE II (DOG).

Heart rate.	Beginning of *Q* to 1st sound.	Beginning of *R* to 1st sound.	End of *T* to 2nd sound.
131	0·017	0·010 or less	—0·016
136	No *Q*	0·004	—0·051
121	0·024	0·018	0·023
130	0·029	0·027	—0·011

The measurements of Table I are in general agreement with those of other observers. The 1st sound begins in man from 0·009 to 0·039 of a second after the deflection *R* begins. The relation of the end of *T* to the 2nd sound is variable; it may fall 0·03 of a second before or after the sound, or in an intermediate position.

* The — sign indicates that the 2nd sound precedes *T*.

If we take the upstroke of *R* and the end of *T* as evidences of the beginning and end of systole, the error in the former will probably not exceed 0·02 of a second and in the latter will not exceed 0·03 of a second.

That *P* constitutes the auricular representative in the electrocardiogram is clearly shown by its time-relations to curves taken directly from the auricular muscle and by its occurrence whenever the auricle contracts, whether the ventricle responds or not (see Chapter XIII on heart-block). The deflections *Q*, *R*, *S*, are known to correspond to the initial processes of the ventricular contraction because they are related to this contraction in time, and because they are to be recorded when the ventricle contracts, whether the contraction follows an auricular contraction or not. The last evidence is to be emphasised in respect of *Q*; it is beyond question a ventricular event, occurring as it does in curves of complete dissociation of auricle and ventricle as part of the ventricular complex, as was first pointed out by Einthoven.

Certain principles of interpretation.

Leading directly from the muscle.—When a simple strip of muscle is stimulated at one end, a wave of contraction passes from the proximal or stimulated end to the distal end. Associated with this wave of contraction there is what is termed a wave of excitation, and this follows the same course as the contraction wave, but actually precedes contraction by a very brief time interval. It is this wave of excitation which is responsible for the electrical changes as we record them.

If a strip of muscle (Fig. 29, *P-D*) is connected to a galvanometer by means of non-polarisable electrodes in contact with its two ends, and if the muscle is then stimulated at *P*, the galvanometer exhibits two deflections. These deflections when recorded form a *diphasic curve*; they are produced by what are sometimes termed "action currents." The first deflection is associated with activity of the muscle strip beneath the proximal contact; the second deflection, which is of opposite sign, is associated with activity under the distal contact. The first deflection has the same direction as one produced when the proximal contact is placed on the zinc, and the distal contact on the copper of a copper-zinc couple. *When muscle passes into a state of activity it becomes relatively negative to inactive muscle*, in the same sense that the zinc of a battery is negative to the copper. This electrical change in the muscle induces a current which passes through the galvanometer from the inactive (+) to the active point (—), and the instrument records the passage of the current by a movement which in our photographs is upward. As the excitation wave travels along the muscle from *P* to *D*, sooner or later it reaches *D* and subsides at *P*;* thus, *D*

* According to the length of the strip and the duration of the active phase, activity at *P* subsides or begins to subside before or after activity is awakened at *D*. The diagram shows the second time-relation, for that is the relation which obtains in the heart.

eventually becomes more active than *P*, and therefore relatively negative to it. The swing of the galvanometer is reversed as a consequence, the current flowing through it in the reverse of the original direction. The two phases of the curve are due to a change in the relation of the active point to the contacts as the wave travels from one end of the muscle to the other. The culmination or summit of the first deflection can be shown theoretically and by demonstration to coincide with the earliest arrival of the excitation process under the distal contact (Fig. 29*b*), for that is the instant at which electrical balance between the two ends of the muscle begins to be restored; a little later, when both ends of the muscle are equally active (Fig. 29*c*),

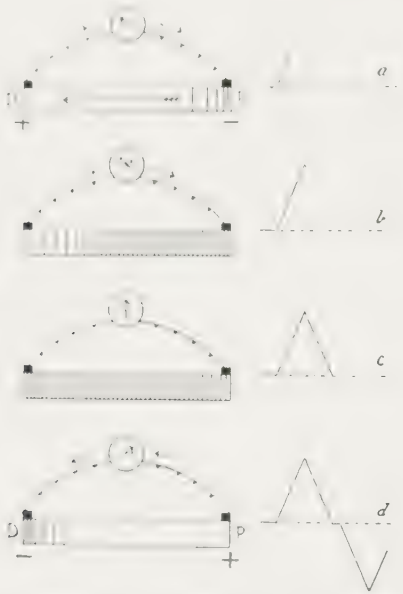


Fig. 29. A simple strip of muscle is stimulated at its right-hand end (*P*). The diagram shows (at *d*) the resultant curve and relates its phases to the events in the muscle.

the two ends of the muscle are *isoelectric*; at this stage no current flows through the galvanometer and the first deflection has been completed. There follows a variable period of equal activity, the isoelectric condition continuing. As activity begins to subside at the proximal end, the second deflection commences, the current flow through the galvanometer being reversed, and is completed as the muscle becomes quiescent (Fig. 29*d*). Now this experiment is a simple one and is readily understood, once it is known that active muscle is relatively negative to inactive muscle. It is fundamental, nevertheless: interpretations of curves taken from the heart are largely based upon it, and in the next chapter we shall see more particularly how it is applied in direct examinations of the heart in experiment.

This first law leads us to a second, which has a wide application. It is that *the direction, which the excitation wave takes* in travelling, governs the form of the corresponding curve.* This second law in so far as it applies to direct leads may be illustrated by means of the same muscle strip, for if the latter is stimulated at *D* and the contraction wave is forced to travel from *D* to *P*, a diphasic curve is still obtained, but the phases as compared to those of the first curve are reversed (Fig 30*b*). The reversal must clearly occur, seeing that the order in which the ends of the muscle are activated is reversed. In interpreting abnormal electrocardiograms, which are taken by means of indirect leads, the known relation between the form of the curve and the direction taken by the wave is also constantly applied. A change in the direction of the contraction wave usually implies that the wave starts from an abnormal point; thus, electrocardiography may acquaint us with the points from which the heart is excited to contract.

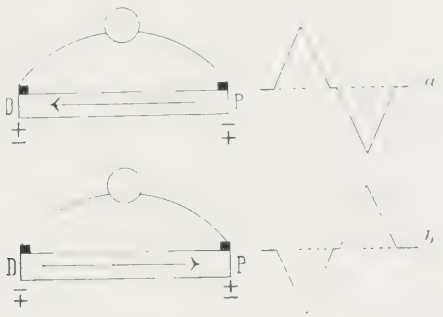


Fig. 30. A simple strip of muscle is stimulated at its right (*a*) or left end (*b*). The diagram shows the resultant curves reversed.

Indirect leads.—In human electrocardiography the electrodes are not in immediate contact with the heart muscle, but with the limbs, and through these with the thoracic tissues. The leads are indirect, and this fact is constantly to be remembered.†

A lead from the right arm to left leg in human electrocardiography is by no means equivalent to a lead from contacts placed in experiments on the actual base and apex of the heart. In human work the contacts with the heart are large; they are constituted by the tissues surrounding the heart on all sides. When small electrodes are placed directly upon the exposed muscle the resultant curve chiefly expresses the events in those small sections of muscle which lie immediately in contact with the electrodes. In human work, in which the contacts are broad, the resultant curves are composite and represent much more in their due proportions the activity effects of all the muscle of the chambers.

* Relative to the leading-off electrodes.

† It may be emphasised by employing Samojloff's terminology (*682*), speaking of curves taken from indirect leads as *electrocardiograms* and direct leads as *electrograms*.

Those who regard a lead from the right arm and left leg in the human subject as a simple base to apex lead, assume that an upright deflection indicates relative negativity of that portion of the whole *chamber* which is nearest to the arm contact (*i.e.*, where the ventricle is responsible for the deflection, the base of that chamber). This assumption is seriously open to question or misinterpretation. In the case of an upright deflection in a human electrocardiogram, it is more correct to assume that *that muscular section of the chamber to which a single excitation wave is confined is so placed that the first part of it to be activated lies nearest to the arm contact.* Thus, where the ventricle is concerned, it may perhaps be agreed to consider the point first activated as equivalent to *P* in Fig. 29. The usual physiological conception considers the whole of the remaining muscle of the chamber as equivalent to *D* from

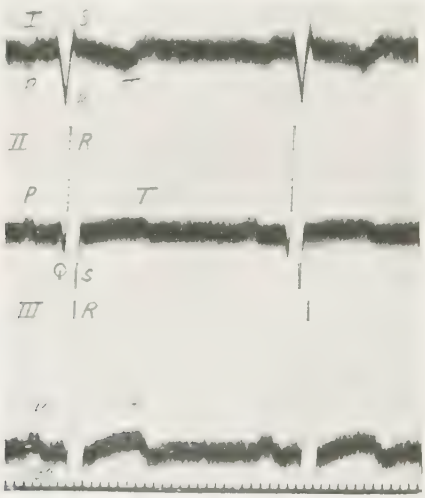


Fig. 31. Electrocardiograms from the three leads in a case of transposed viscera. All the deflections of lead *I* are inverted; the deflections of the remaining leads are natural in direction. Time-marker in thirtieths of a second.

the very first. This conception is rendered difficult, if indeed it is accurate, for, as we shall see, the excitation wave starts almost simultaneously at a large number of points. If a given excitation wave starts at a point in one small section of the ventricular muscle, that point may be considered as equivalent to *P*; *D*, to my mind, is not represented by the remainder of the ventricular muscle but by that section of it through which this particular excitation wave will travel. The excitation wave in question has a limited territory, at the imaginary boundaries of which it meets neighbouring excitation waves, and meeting them is unable to pass further. Thus it would be a matter of indifference whether the section of muscle lies near the base or apex of the ventricle, providing that that part of it which first becomes negative lies nearer the right shoulder, and that part of it which becomes

negative a little later lies nearer the left thigh; such deflection as is seen during the spread of the excitation wave will be upright. In other words, if the direction in which the excitation wave travels is, on the whole, away from the arm contact and towards the leg contact, the resultant deflection will, by its uprightness, proclaim the fact.

The manner of ascertaining the more precise direction in which the excitation wave travels by means of the indirect leads of human electrocardiography will be described in a later chapter when the occasion arises.

For the moment we may consider the broader question, that the electrocardiogram indicates the course taken by the excitation wave as a whole. A simple clinical demonstration first accomplished by Waller (*745*) will suffice to show that the curve obtained from the heart is controlled by the direction taken by the excitation wave in its relation to the leading-off contacts. When the heart is normal and normally situated in the body, the deflections *P*, *R* and *T*, are upright in direction (see Fig. 21) in curves taken from lead *I* (right arm to left arm). But when a patient who presents transposition of the heart is similarly examined, the direction of all deflections *in this lead* is inverted (*330*, *574*) (Fig. 31). The differences in potential between the right arm and left arm in a normal subject, during the progress of the heart beat, are precisely the same as the differences of potential between the left arm and the right arm in a dextrocardiac subject. A little reflection will show (as does Fig. 31) that this inversion will occur only in lead *I*, for lead *I* is the only symmetrical lead of the three used (*583*, *683*). Inversion of all the deflections in lead *I* in the absence of reversal in the other leads is a reliable physical sign and probably the most reliable sign which we possess of dextrocardia.

Chapter V.

The Normal Pacemaker of the Mammalian Heart.

In the hearts of the cold-blooded vertebrates, the great veins, systemic and pulmonary, terminate in the first chamber, the *sinus venosus* and the sinus and the true auricle into which it leads are clearly defined. Consequently it is a matter of little difficulty to show that the heart beat arises as high up as the line of union in these animals. A ligature or clamp placed upon the sino-auricular junction (1st Stannius ligature), thereby isolating the sinus, brings the lower chamber to a condition of standstill, while the upper portion of the dissociated musculature preserves its original rhythm. In the mammalian heart no additional chamber, in the form of a sinus, exists. It is natural to seek the remains of sinus tissue at those points at which the great veins enter the heart, and for many years the *sulcus terminalis*, a groove on the outer surface of the auricle, has been regarded as the line separating the representatives of the two chambers. Earlier workers were content to differentiate two portions of the auricle, one above and one below this line, and their experience, coloured as it was by contemporary morphology, spoke simply of a pacemaker in the region of the great veins. But recent anatomical research has sharpened our knowledge. Keith and Flack hold that there are sinus remnants in the region of the mouth of the superior vena cava, in the coronary sinus, in relation to and in the auricular septum, and possibly also at the mouths of the pulmonary veins, for they find masses of differentiated muscular tissue in these situations.

Anatomical and morphological researches led Keith and Flack (*372*) to suspect that the pacemaker lies in one of the collections of specialized tissues found in the auricle, and directed their attention more particularly to the sino-auricular node ; for this is a relatively large collection of peculiar tissue, and it stands in intimate relation to the rich supply of nerves entering the heart in its neighbourhood. The statement of these views has been responsible, directly or indirectly, for the most recent observations. No sooner was it known that a mass of tissue, remarkable for the peculiar form and arrangement of its elements, exists in the neighbourhood of the superior cava, than experimental pathologists turned close attention to this region.

Of the many experimental methods which have been adopted in locating the position of the pacemaker in the mammalian heart, the chief may be summarized. Many of the observations have been upon hearts subjected to considerable mechanical or chemical injury, or upon hearts in which normal nutrition was disturbed. I propose, therefore, to deal first with the electrical methods, which have been undertaken in a manner free from these objections, for they are, to my way of thinking, the most perfect we possess for the purpose and are sufficient to show the region of the auricle which first becomes active.

Forcing an excitation wave to follow the natural path.

It is known that the electrical curve yielded by an unexposed and naturally beating auricle exhibits very great constancy from animal to animal and from one species of animal to the next. We may conclude, therefore, that wherever the pacemaker lies it has a similar position in different animals of the same species, and in animals of separate species (man, horse, pig, dog, cat and rabbit); so that if there are several widely separated centres, from any one of which the excitation wave might be supposed to originate, it does not spring from this in one animal and from that in another. If we are satisfied that the sino-auricular node is the pacemaker in the dog, we shall be satisfied that it is the pacemaker in all mammals in which this node is present, and in which the heart yields the same type of auricular electric curve in a given lead. Now auricular contractions may be excited from any desired part of the auricular musculature by means of single induction shocks, and such experiments may be performed under extremely favourable conditions, with natural respiration, with the thorax closed and with the heart beating at a normal rate (*445*). Successive excitation of many points of the auricular musculature (right and left) shows with certainty that the auricular electric complex obtained when a heart beat is excited from the neighbourhood of the superior vena cava precisely resembles the normal auricular complex. The meaning of this fact is clear, for the form of the electric curve indicates the direction of spread. The path taken by the normal wave in the auricle is similar to that taken by a wave excited artificially from an area surrounding the mouth of the superior vena cava; in other words, the normal and the excited waves are propagated from the same area. It is also found that auricular curves of a similar nature are yielded by excitation of no other area, but that in any given animal the type of curve obtained from inferior vena cava, coronary sinus, pulmonary veins, etc., bears no resemblance to the normal auricular curve.* These facts are illustrated in Fig. 32.

* Rothberger and Winterberg state their belief that the curves of these experiments of mine were materially complicated by the break shocks of stimulation (*665*, p. 601). This was certainly not the case. Such deformity of the curves is usually avoided if threshold stimuli are employed, and if the electrodes are close together. Any deformity arising from this source is easily recognised and allowed for, and should but rarely occur.

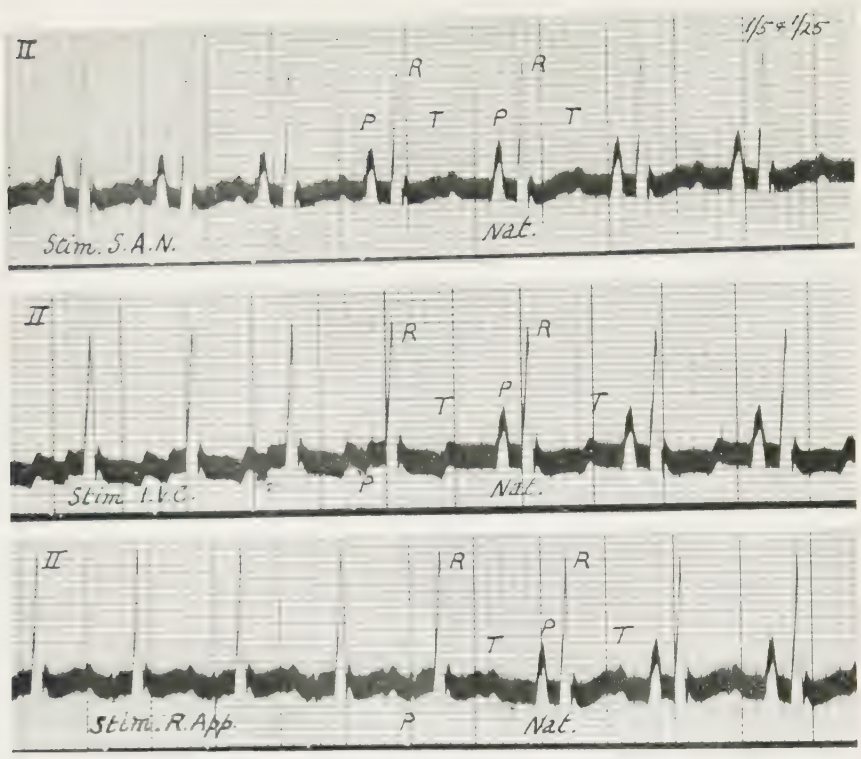


Fig. 32. Three electrocardiograms from a dog, each taken by lead *II*. The last three cycles in each strip of curve correspond to natural heart beats. The early cycles of each strip are responses to stimulation in the region of the sino-auricular node (*S. A. N.*), the inferior vena cava (*I.V.C.*), and the tip of the right auricular appendix (*R. App.*). In each instance the forced beats resemble the normal beats in so far as the ventricular elements (*R* and *T*) of the curve are concerned, but only when the beats are forced from the region of the sino-auricular node are the auricular elements (*P*) the same for forced and normal beats. Time in fifths and twenty-fifths of a second.

The point of primary negativity estimated by the direction of deflections.

We have seen that when a strip of muscle becomes active at a given point, this point becomes negative relatively to all other points. This law has been familiar since its statement by Hermann. Negativity is readily shown by a galvanometer, for, if the recording instrument is connected by means of non-polarisable electrodes* to two points upon the surface of a muscle, primary negativity of one contact is shown by a deflection of the

* These electrodes consist of small glass tubes plugged with salted kaolin and half filled with saturated copper sulphate solution in which copper wires are immersed. The contact with the surface of the heart is effected by means of cotton threads smeared with kaolin and imbedded at one end in the kaolin plug.

string in a known direction. This principle permits us to test our hypothesis that the first active point of the auricular muscle is in the region of the *S-A* node. If one contact is placed over this region and the other is placed successively upon points lying around it, then, if activity is always first developed under the central contact, this contact should always become primarily negative to the outlying contact. That is to say, if such radiating leads are taken, maintaining the central contact upon the point at which the excitation waves arises (Fig. 33), a series of electrograms should be obtained, in each of which the first deflection is in a given direction ; the direction of this deflection ought always to indicate primary negativity of the central point.

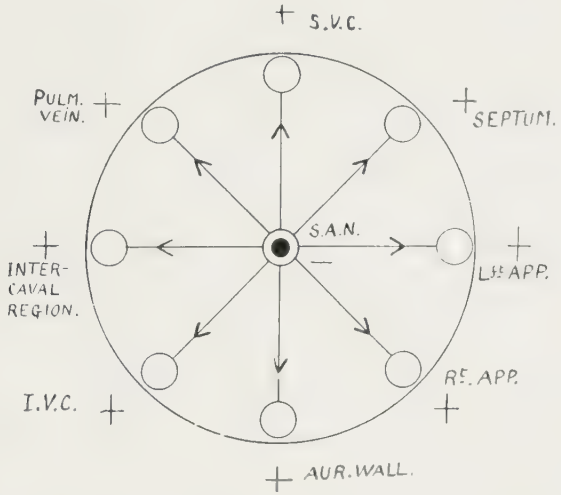


Fig. 33. A diagram illustrating the electrical condition of the *S-A* node at the beginning of auricular activity. The muscle in this region becomes relatively negative to all other points on the auricular surface.

Now there is only one superficial region of the mammalian auricle which exhibits these electrical relations when the heart is acting normally. If one contact* is placed over the region of the sino-auricular node and the other contact is moved to any other point on the auricular surface, it matters not where the second contact is placed, the first deflection obtained with auricular systole is upward in direction and indicates relative negativity of the centre point. A diagram illustrating the contacts in an actual experiment is shown in Fig. 34. Examples of the electrograms are shown in Fig. 35.

* That which gives, when connected to the zinc terminal of a copper-zinc couple, an upright deflection.

This type of experiment goes a long way to convince, for the muscle of the auricular wall is thin and the *S-A* node lies in what may be regarded as the centre of a muscle sheet, that is to say, points may be chosen for examination all around it. There is little or no chance of this region receiving the excitation wave from a deeper structure, for all the muscle bands running towards the node may be tested. The conclusion that it is the centre in which the excitation wave starts is strongly suggested by these observations, which were first undertaken by Wybauw (*794*, *795*), and in my own laboratory (*440*, *485*), nine years ago. Since the original reports appeared,

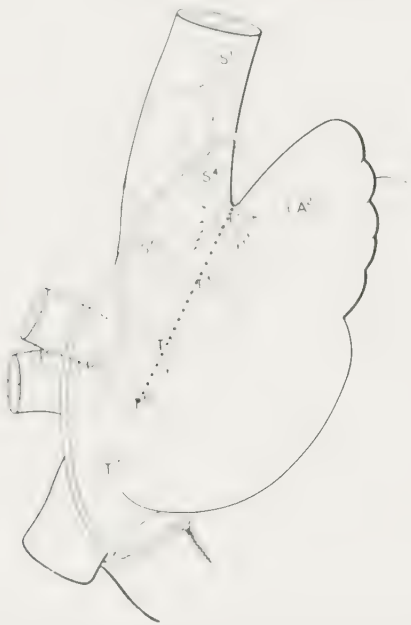


Fig. 34. (*Heart*, *1910-11*, *11*, *158*, *Fig. 4.*) A diagram of a dog's right auricle showing a series of paired contacts. The contact T^2 was maintained at the cephalic end of the *sulcus terminalis*, the contact paired with it was moved to different points of the surface of the auricles and great veins. In each instance the first deflection corresponding to auricular activity was upright, indicating primary negativity of T^2. The general direction of spread of the excitation process, as indicated by this method, is shown by means of arrows. The distribution of the sino-auricular node, ascertained histologically, is shown by the dotted line.

I have repeatedly confirmed them; they have also received recent confirmation by Eyster and Meek (*161*).

In the pig, the sino-auricular node lies higher up the sulcus than in the dog, and in one animal of this species examined the point of relative negativity was found in a corresponding position (*464*). It lay, as Dr. Ivy Mackenzie was subsequently able to show histologically, immediately over the sino-auricular node in this animal, as it had lain in all our experiments upon dogs.

Extrinsic and intrinsic deflections.

Before pursuing our discussion, it is desirable to consider what may be termed "outlying" leads—leads in which neither contact lies over the head of the *S-A* node. Such a lead is illustrated in Fig. 34, S^4-S^1. In leading directly from the heart muscle, the chief deflections occur when the excitatory process is produced or arrives immediately beneath the contacts; the contacts are exposed to the full force of the electric discharge in the active tissue underlying them. Such leads are essentially different from those used in human electrocardiography, for in these the electrode contacts are upon the limbs. Curves of the excitation wave may

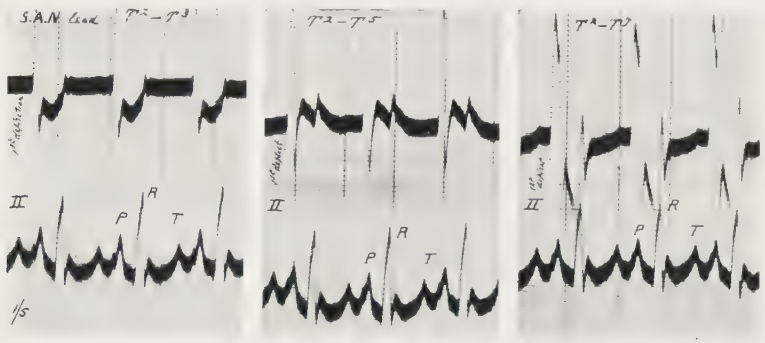


Fig. 35. ($\times \frac{3}{5}$.) Three photographs, each showing a simultaneous electrogram and electrocardiogram in a dog. In each photograph the lower curve was taken by a constant lead (lead *II*). The upper curves (or electrograms) were taken directly from the exposed auricle of the dog. In the left hand figure the contacts both lay near the cephalic end of the *S-A* node (T^2 to T^3 in Fig. 34). In the middle figure the upper contact was maintained (T^2), the lower being placed near the midpoint of the sulcus (T^5). In the right hand curve the upper contact was maintained at T^2, the lower being placed at T^7 on the inferior vena cava. In each curve of the direct leads the first deflection starts sharply and is prominent and upright. Each of these deflections is almost simultaneous with the beginning of *P* in lead *II*. At the beginning of the auricular systole, therefore, the region of the *S-A* node was primarily negative to the three other points on the sulcus. Time in fifths of a second.

be obtained by both methods, namely, when contact with the active structure is direct or when it is indirect through inactive tissue, and the electric effects of the two orders should be distinguished. Now, when contacts are placed on the muscle of the auricle they produce deflections of two kinds (*483*). The chief deflections are those which result from the arrival of the excitation process immediately beneath the contacts; these are termed *intrinsic*. They are deflections, which represent relatively large electrical potentials and they have correspondingly large amplitudes.* The deflections of the

* Exceptionally the intrinsic deflection is not the most prominent in the electrogram.

second order are those yielded by the excitation wave travelling in distant areas of muscle. These are qualified by the adjective *extrinsic*. Intrinsic and extrinsic deflections may be illustrated by a simple experiment. If two contacts are placed upon the sulcus terminalis, then at each beat of the auricle a large intrinsic deflection is produced as the auricular excitation wave reaches one contact. But the same contacts also record the subsequent discharge of the ventricle. These last effects are extrinsic, representing the activity of distant muscle elements. A similar double effect is noticed in respect of the auricle itself. If we lead from two contacts lying over the right auricular appendix, an example of an outlying lead, we obtain a curve

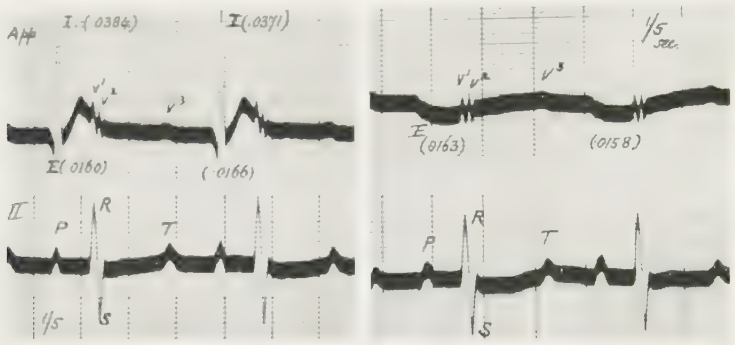


Fig. 36*a* and *b*. (×⅔.) Simultaneous electrogram and electrocardiogram in a dog. The upper curves are from the auricular appendix, the lower curves are from lead *II*. To show the effect of crushing the base of the appendix and rendering the tissue under the contacts inactive (*a* before, and *b* after, the crush). The chief or intrinsic deflection (*I*) is abolished by the crush; the extrinsic auricular deflection (*E*) remains, as do the extrinsic ventricular deflections (V^1, V^2 and V^3). That *E* is the same deflection in both figures is shown by its direction and by exact measurement; it begins in both curves approximately 0·016 of a second before the summit of *P* is inscribed by lead *II*. Time in fifths of a second.

(Fig. 36*a*) differing in form from the curves where one contact lies over the *S-A* node (Fig. 35) in an important respect. If we examine the upper curve (or electrogram) in Fig. 36*a*, we see the usual tall spike (*I*), but it is preceded by a small dip in the curve (*E*). This initial dip is not produced by activity in the appendix, on the other hand the tall spike is; these facts are readily demonstrated by crushing the base of the appendix. By this procedure, the appendix, though uninjured beneath the contacts, is rendered inactive, and as soon as this has been effected the type of curve changes. The extrinsic or small initial deflection remains, while the intrinsic effects disappear (upper curve, Fig. 36*b*). Now this demonstration is fundamental, for it permits the analysis of those curves which are obtained from outlying leads. Almost all such leads give curves of composite form; consisting of a main deflection, corresponding to the arrival of the excitation process

beneath the contacts, and initial deflections which are due to the passage of the excitation process through neighbouring or distant masses of the tissue. In these curves the *main* deflection is of the same nature as the first deflection when the lead is from the *S-A* node. In considering the course of the excitation wave, as opposed to its origin, attention should focus therefore upon these chief or intrinsic deflections.

The point of primary negativity estimated by timing the excitation wave.

The last experiment has been described to show that, in a curve taken by a direct lead, it is possible to identify the deflection, which represents the arrival of the excitation wave beneath the contacts. If direct leads are employed simultaneously with a constant lead such as lead *II* (Fig. 37), the times at which the excitation wave appears at different points of the auricular muscle may be ascertained relative to each other. For this purpose very accurate mensuration is required, and it is essential that the curves which are to be magnified for measurement by the comparator (Fig. 19, page 41) should be perfectly clean cut. When the experiments are performed in an exacting fashion, it is possible to reduce the customary error of measurement to less than a thousandth of a second, and to obtain a series of readings for points on the surface of the auricle, to relate them accurately to each other and thus to ascertain the order in which they become active. The whole superficies of both auricles and the septum internally has been explored in a large number of animals by this method (*483*), and it can be affirmed that the first appearance of the excitation wave is over the head of the sino-auricular node and that it appears at later times in all other regions of both auricles, while the heart beats naturally.*

The same observations provide this and another important evidence that the excitation wave begins in the region of the *S-A* node. It is the only region of the auricle, from which curves are obtained, which uniformly yields no initial deflections (as at *E* in Fig. 36*a*) ; the reason being that when the intrinsic deflection is obtained from this lead, the whole of the rest of the auricular tissue is at rest ; while in all outlying leads the intrinsic deflection is preceded by initial movements of the string which represent currents from the preceding activity of the *S-A* nodal region and its surrounding areas (see Fig. 37). Thus there is abundant and conclusive evidence that the excitation wave commences in or near the head of the sino-auricular node.

This conclusion prompts a second, for if the excitation wave first appears in the region named, some tissue, there situate, probably provokes the heart beat. The next experiments relate to this question.

* Sulze (*715*) also finds by a similar method that the excitation wave is first shown by the nodal region as opposed to the remaining superficies of the right auricle.

Warming and cooling.

It is wise to seek knowledge of the normal heart beat from a naturally suspended and naturally nourished organ. The method now described fulfils these conditions. Entailing, as it does, minimal disturbance, it ranks as one of the most important ways in which the site of normal impulse formation may be sought. The method is based upon the general observation that, while a rise of temperature increases, a fall of temperature decreases the functional activity of the tissue to which it is applied, and upon the more particular observations of Gaskell and Engelmann, who found an increase or decrease of the whole heart rate in the frog when the sinus was submitted to heat or cold, respectively.

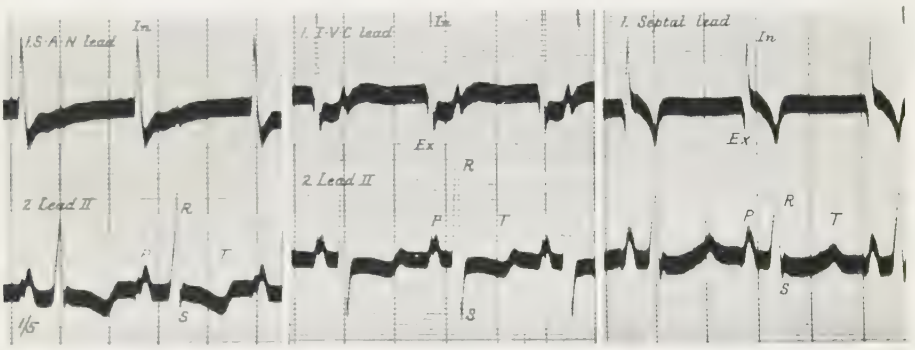


Fig. 37. (×⅘.) Simultaneous electrogram and electrocardiogram from a dog. The upper curves (or electrograms) were obtained from the region of the *S-A* node, the inferior vena cava and the auricular septum respectively; the lower curves, forming the standards of time measurement, are from lead *II* in each instance. The three illustrations are from separate animals. Time in fifths of a second.

In the mammalian heart, the first experiments were undertaken by McWilliam (*523*), who states that the "application of slight heat locally to the terminal part of the vena cava superior gives a marked acceleration in the rhythm of the whole heart." Adam (*2*), using the excised heart for the most part, made more extensive experiments, and stated that the point most sensitive to temperature change lies between the superior and inferior venous inlets, and rather nearer to the latter than to the former.

The first experiments in which account was taken of the position of the *S-A* node were reported by Flack (*173*). He states that the application of cold immediately slows the whole heart, but that this effect is obtained only when the temperature is lowered over the region of the node.

A thorough investigation has been undertaken lately by Ganter and Zahn (*203*, *204*). These workers employed exact methods and checked their experiments histologically. They used a large number of various animals, experimenting upon the heart beating *in situ*. In cats, goats, dogs and apes, their results were uniform. Cooling the region of the node produced slowing, warming produced acceleration of the whole heart. These

effects were obtained only when those portions of the superficies were tested which were close to nodal tissue. The whole length of the node was found to be sensitive, *but the further the point investigated lay from the head of the node the less were the effects.*

These observations are important. The method employed is one of extreme delicacy; the results are easy to confirm and are conspicuous, not only for their uniformity, but for the exact correspondence between the area sensitive to temperature and that which overlies the nodal tissue. The experiments corroborate those in which the area of primary negativity has been sought. Both methods indicate that the upper end of the node is its most active part. But the method of cooling goes further, for it shows that the *impulses originate* in this heart region. Simultaneous observations were made by Brandenburg and Hoffmann (*40*, *41*); they report experiments upon the effects of cooling in the excised and perfused mammalian heart. Their results are in full accord with those already quoted.

Other observations.

Destruction of the sino-auricular node.—In attempting to throw the sino-auricular node out of action different workers have utilised different methods. To avoid the hæmorrhage consequent upon breaking the heart wall by incision some have adopted perfusion (*70*, *71*, *530*, *561*). Now all such experiments are open to criticism; the heart is an extremely sensitive structure, it is intolerant of injury, and especially it is intolerant of nutritional disturbance. The effects of perfusion have always to be considered in weighing the results of experiments performed by its means. There have been three series of observations upon the perfused heart.* Magnus-Alsleben (*530*) used rabbits and found very little disturbance when the region judged to contain the node was excised; he failed to bring forward clear evidence that the node had been removed in part or in whole. Cohn and Kessel (*70*, *71*) employed dogs, and in a large series of experiments obtained effects which were positive and very fairly uniform. Their plan was to make the first three cuts of a rectangle, which when completed would surround the node, and thus to leave the node connected to the rest of the auricle by a bridge. The result of this procedure was negative, but the fourth and final incision produced disturbance, provided that the node was effectually circumcised. They state that excision of the node results in an immediate standstill of the whole heart, while excision of the

*It seems almost unnecessary, at this time of day, to do more than refer to the earlier experiments of Langendorff and Leymann (*408*), Hering (*273*, *302*), and Lohmann (*493*). The first attempted the separation of the remaining portions of the heart from the complete venous inlet. The amputated portions demonstrated standstill, while the stump continued its contractions. Hering made cuts in the neighbourhood of the superior vena cava and obtained standstill. Lohmann destroyed the tissues of the inlet by applying formalin to them, and obtained dislocation of the centre of impulse formation as a result.

neighbouring portions of the auricle fails to produce this effect. Slowing or standstill is shown by their protocols, which include accounts of the microscopic findings.

Moorhouse (*561*) employed the dog's heart and found that removal of an area containing the upper half of the sulcus terminalis produced variable results. There might be standstill of the auricles and a subsequent assumption of a relatively slow rate, a gradual slowing of the auricle, or no change. He also saw the same effects after removing the lower half of the sulcus terminalis. In some instances, at all events, histological observations were undertaken.

These results are therefore in apparent conflict; in one series of experiments excision of the perfused node gives uniform standstill or slowing, in another these effects are but occasional, while in another they do not occur.

Several experimenters have utilized *the intact heart beating in situ*, and have sought to destroy the node by burning it. Both Jaeger (*342*, *343*) and Hering (*302*) applied a cautery to the auricle. Again the results have varied, for while Jaeger, who used dogs and cats, found no slowing after very extensive destruction of tissue,* Hering, who used dogs chiefly, noticed alterations in the sequence of contraction, indicating a dislocation of the site of impulse formation, after far less extensive destruction. Flack (*174*, *176*) excised the area of the node, or used a clamp to isolate the node in dogs and in rabbits; he found little change in rate, or only slight slowing.

The disagreement amongst workers, using either excision or destruction by cautery, convinces us that their problem is no simple one, but that the result is controlled by the interplay of several factors. And there are two factors of consequence. In the first place, injury in the region of the sulcus interferes with the special arterial circle which lies in this sulcus and supplies the node. Secondly, the centres having a function of rhythmicity are extensive; when one centre is destroyed, or, if nutrition is imperfect, before it is destroyed, another centre may usurp its function (see Chapter XV); in some series of experiments this process of substitution may occur more readily than in others. That several areas of the auricular substance are capable of promoting rhythmic impulses has been shown by the observations of Erlanger and Blackmann (*145*, *147*).†

In the hope of bringing some of these conflicting experiments into line, I undertook a series of experiments upon cats (*464*). Using the heart beating *in situ*, I clamped the area containing the upper third or half of the sulcus, and by pressure destroyed the tissue in this region. In twelve

* Extensive burning, such as Jaeger employed, is a crude experimental method.

† This is generally admitted, though there is still doubt as to whether such rhythm producing areas do or do not contain portions of the nodal tissues (see page 204 footnote and context).

experiments I obtained slowing of the heart on all occasions, which in all but two experiments was permanent. The fall of rate varied from six to fifty beats per minute. Subsequent microscopic examination has sometimes shown destruction of the node, but often the node has escaped. These observations confirm those of Moorhouse in that slowing may be obtained when the node lies outside the crushed area, but to what extent it may have been damaged by the crush in its neighbourhood it has often been impossible to tell.

What is almost constant is a change (temporary or permanent) in the type of the auricular portion of the electrocardiogram, whenever the clamp includes the sino-auricular node; and often when the crushed area

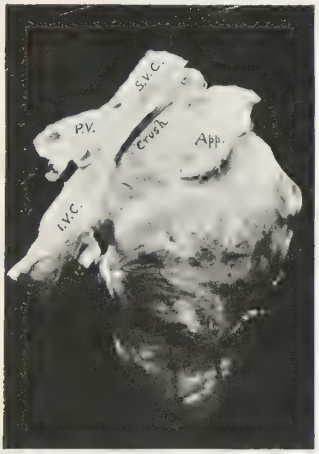


Fig. 38.

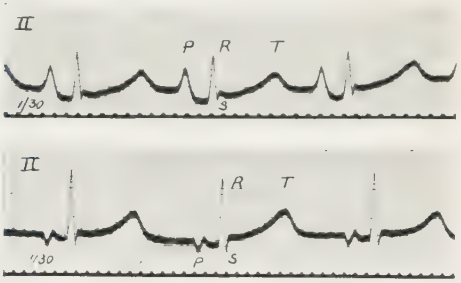


Fig. 39.

Fig. 38. Photograph of a cat's heart, regarded from the right side. At the base of the crinkled appendix the crushed tissues are raised in a line along the course of the sulcus terminalis.

Fig. 39. ($\times \frac{4}{5}$.) Two electrocardiograms from this animal, taken immediately before and after crushing the region of the *S-A* node. In the lower figure the rate is slower, and the auricular summit, *P*, instead of being upright, is now inverted, indicating an altered seat of impulse formation. Time in thirtieths of a second.

is simply close to it, the auricular summit vanishes or becomes inverted, indicating a change of the seat of impulse formation (Fig. 39). If the node has not actually been included, then the original impulse centre may re-awaken during later stages of the experiment. I have been unable to obtain these effects from other portions of the right auricle. But I lay no stress upon the results. Upon the other hand, the observations have taught me to be sceptical of the whole class of experiments; they are beset with pitfalls.

The difficulties with which we have to deal in attempting to destroy the node *in situ* and in observing the after-effects, are numerous. It is rarely possible to estimate the degree of damage to the node, unless a large area of the auricle is completely destroyed. If any portion of the node remains, or if the whole is left, we cannot determine the degree of its functional activity. It is often quite impossible to be certain whether small portions of the node remain or, if ascertained to be remaining, whether these are functionally intact or not.

OBSERVATIONS ON THE DYING HEART, ETC.

(*a*) *The ultimum moriens.* In the moribund amphibian heart the last portion of the musculature in which contractions are visible is the sinus. A number of parallel observations has been undertaken upon the mammalian heart, and these date back to the times of Harvey and Albrecht von Haller. It has been stated that the contractions are last observed in the terminations of the great veins (*382, 523*); for example, in the superior vena cava (*273, 279*). On the other hand, they have been observed in the auricular appendix, in the pulmonary veins, and other parts of the heart, such as the auriculo-ventricular junction. The observations are coarse and probably largely unreliable. As McWilliam (*523*) stated thirty years ago, it does not follow that the origin and sequence of the normal heart is similar to that in the dying organ (see Eyster and Meek's recent paper (*162*)).

(*b*) The beats of the dying heart are slowly conducted, and the contraction-waves may often be seen to originate near the mouths of the great veins and to be conducted from them (*279, 523*). Observations upon the dying heart cannot be held to have any great weight in deciding the site of the pacemaker. The heart is in a pronouncedly abnormal state, and it is well known that small nutritional disturbances may readily dislocate the seat of impulse formation.

(*c*) *Method of forcing extrasystoles.* This method of searching for the pacemaker by examining the lengths of the pauses following upon the forced beats can no longer be regarded as carrying much weight, though its results are actually in accord with our knowledge of where the pacemaker lies. (See page 224.)

NOTE.—For recent observations on the pacemaker in the reptilian heart the papers of Meek and Eyster (*549, 552*), and for similar observations on the avian heart the papers of Flack (*175*) and of Mangold and Kato (*532*), may be consulted.

Chapter VI.

Function of the *A-V* bundle and curves resulting from lesions of its two divisions.

In many organs, where a contraction is propagated as a wave from point to point, transmission can be shown to depend upon the functional integrity of the tissues directly uniting such points. The experiments of Romanes (*657*) upon the umbrella of the jelly fish, in which such conduction first received careful study, laid the foundation of our present knowledge of this subject. His researches were followed by those of Gaskell (*215*) upon the amphibian and reptilian heart. The detailed observations of Gaskell exhibited the dependence of conduction upon direct muscular continuity, and presented a clear conception of hindered conduction (heart-block) in the cold-blooded heart.

The final steps, the demonstration of similar phenomena in the mammalian heart, followed upon this work. Amongst the earliest experiments were those of Tigerstedt (*728*), Wooldridge (*793*), and McWilliam (*523*); these investigators were able to prove that the normal ventricular rhythm is subservient to impulses received from the preceding contractions of the auricle.

The evidence that the auriculo-ventricular bundle transmits the impulses from auricle to ventricle in the mammalian heart.

Disturbed sequence of contraction in the chambers of the mammalian heart has been recognised for many centuries, though the cause of it remained obscure for a long while. Harvey, in his essay on the movements of the heart, stated that he noticed a prolongation of the interval between the auricular and ventricular systole, and observed an occasional ventricular contraction following upon several auricular contractions in certain circumstances. The earliest attempts artificially to dissociate the auricle and ventricle have nowadays little more than a historical value. They were successful, but involved widespread damage to the heart. Two years after the publication in which His described the auriculo-ventricular bundle, the same observer (*318*, *319*) attempted, in conjunction with Graupner, to destroy the connection between auricle and ventricle in the heart of a rabbit, and apparently succeeded, for he describes an experiment in which

dissociation of the auricular and ventricular rhythm was produced (1895); he took no graphic records, neither did he examine the lesion with the microscope.

In 1904 Humblet (*334*) operated upon the heart *in situ*. Having tied the vena azygos and obstructed the inferior and the superior vena cava, he opened the right auricle and damaged the region of the bundle, subsequently closing the wound in the auricle and restoring the circulation. The method proved unsuccessful in his hands, and he discarded it in favour of perfusion, using the isolated heart. Dogs were employed. He reported that cuts in the neighbourhood of the bundle were without effect, but that when the bundle was damaged dissociation appeared. There is no mention of subsequent histological examination of the bundles, and the single tracing which is given is unsatisfactory.*

At a later date Humblet (*335*) undertook further observations upon the dog's heart. The ventricle of the excised heart was nourished by transfusion; ligatures were passed around the bundle and drawn tight. Eight experiments yielded seven definite results; in each case dissociation was obtained. Curves illustrating the abnormal mechanism were given from three of the animals and also drawings of the lesions as they appeared microscopically. In the eighth experiment heart-block appeared spontaneously.

Hering's results (*262*, *264*, *265*, *266*, *271*) were published in 1905-6. The bundle was damaged in the heart of four dogs. In three instances, in which its tissue was entirely transected, complete heart-block occurred (curves from two of these animals have been published). In the fourth animal the bundle was only partially divided, and in this instance it is stated that no heart-block manifested itself. The extent of the lesions was ascertained histologically (*718*). Hering also demonstrated that lesions which break the anatomical continuity of auricle and ventricle at points other than that at which the bundle is found do not lead to dissociation.

In the same years Erlanger (*142*, *143*) published his first observations; he attempted to pass ligatures through the heart beating *in situ*, in such a way as to ensnare the bundle. In seven experiments heart-block was seen once. The method was abandoned. In a second series of experiments an auriculo-ventricular clamp was utilised. Seven experiments yielded heart-block on two occasions. Eventually he employed a specially-devised clamp, the lower blade of which consisted of a long-shafted fish-hook bent at a right angle. Introduced between aorta and pulmonary artery, the point of the fish-hook was carried into the left ventricle and thence through the ventricular septum below the bundle. The second blade rested between the pulmonary artery and aorta externally. The blades of the clamp could be tightened to the desired extent, and the bundle squeezed or actually crushed. In seventeen experiments upon the heart of the dog beating *in situ*, heart-block

* As Fredericq (*191*) has since told us, the auricular and ventricular curves were incorrectly labelled.

was obtained in sixteen instances, and these, according to the histological findings of Retzer, were the experiments in which the bundle had been damaged. The observations have been extended and confirmed by this worker and his collaborators (*148-151*), and also by Tabora (*716*), using a similar method.

Later, Cohn and Trendelenburg (*77, 734*) worked upon the perfused heart, and their work was conspicuous for the number of experiments and the care with which they were performed and reported. In all, the hearts of 26 cats, 17 dogs, 4 rabbits, 2 apes and 4 goats were successfully investigated. In each instance a complete account of the resulting disturbance in the heart has been reported, together with a diagram of the bundle and lesion, reconstructed from serial sections; usually the protocol is accompanied by an actual tracing of the heart's mechanism, frequently by photographs of the lesions themselves. The results of this work, remarkable for the thoroughness of its execution, may be stated in general terms. Where the incisions failed to reach the bundle, or where it was but partially transected, no disturbance, or merely a partial or temporary heart-block, was encountered. Where the bundle was completely transected, complete heart-block invariably ensued.

The experiments considered all lead to the conclusion that the auriculo-ventricular bundle is the essential organ of conduction; its destruction yields a uniform result, namely, complete dissociation of the auricular and ventricular rhythms; while damage to the surrounding structures or to the remaining anatomical connections between one and other chamber are without effect (*77, 143, 271, 335*).

Certain observations from the Berne school have seemed to contradict those already quoted.

Kronecker and Busch (*395*) worked with *rabbits*, and stated that the bundle might be broken without interference with the heart's sequential rhythm. Kronecker (*394*) himself reported similar results in the dog. Imchanitsky (*340*) used the rabbit's and dog's hearts, the organs beating *in situ*, and worked by the method of ligation adopted by Kronecker. In the instance of a rabbit in which no dissociation was obtained, it is stated that the microscopic examination showed destruction of the bundle. It is not stated that serial sections were cut.

Lastly, Paukul (*590*) reports the general results of experiments upon 24 rabbits; he followed Kronecker's method. He concludes that when the bundle is caught in the ligature without much damage to the surrounding structures, no dissociation occurs, and that dissociation may occur when the ligature passes in the neighbourhood of the bundle, leaving it intact. He offers histological drawings and the curves from several animals as evidence.

The following objections may be urged against the conclusions of the Berne workers. In the work of Kronecker and Busch and Kronecker the histological proof of the bundle transection was not given.* The solitary observation of Imchanitsky and the more extensive observations of Paukul, which alone demand serious attention, are accompanied by a report of the histology. But the sections were cut at right angles to the course of the bundle, and it is probable that branches of the bundle leaving the main tract in the earlier part of its course would escape observation under this technique. The argument becomes more weighty when it is known that such early outgoing fibres are usual in the rabbit (*77*).

* A similar objection applies to the observations of Biggs (*33*), and of Cullis and Dixon (*83*), whose conclusions are, however, of an opposite kind; these workers used rabbits,

The experimental work upon the auriculo-ventricular bundle has been given in full, not only because a conclusion which is fundamental to cardiac pathology rests largely upon it, but because the review allows a limited presentation of experimental methods adopted in such heart work.

In summing up, it may be said that in regard to the work upon the dog no doubt remains, and in regard to the experiments upon other animals the results are concordant, with the single exception of the rabbit.

Possessing the reported observations upon this animal alone, we might hesitate to assert that the auriculo-ventricular bundle is the path along which impulses are sent from auricle to ventricle. Considered in conjunction with the overwhelming testimony of experiments upon other species, a definite conclusion may be formed, which is strengthened by the manner in which the bundle distributes itself in the rabbit. It is proved that a limited tract of tissue stretches between auricle and ventricle, and that the co-ordination of auricle and ventricle is dependent upon its integrity. Further, it may be stated that the integrity of the tissues uniting the auricle and ventricle at all other points is immaterial to impulse transmission.

In the clamp experiments of Erlanger and in the section experiments of Cohn and Trendelenburg, it not infrequently happened that when the bundle was damaged to a limited extent the grade of heart-block was partial. In Erlanger's experiments, successive turns of the screw tightening the clamp produced increasing degrees of heart-block, and this investigator obtained results in every way parallel to those obtained from the tortoise heart by Gaskell.

Such disturbances of the heart's rhythm do not follow when one or other division of the bundle is broken (*77*, *137*). They do not necessarily follow when the bundle is partially divided; it is apparent from experiment that conduction may be unimpaired when a partial section is executed, and the same experiments suggest that where partial section occurs and complete dissociation supervenes, it is the result of section of some fibres and damage to the remaining fibres of the conducting track. A strand of the bundle with its two intact branches, or the whole bundle with one or other of its branches, is sufficient, if undamaged, to preserve a conducting track.

Erlanger's experiments (*148*, *151*) upon dogs have shown that a bundle when once destroyed is not repaired. He succeeded in restoring animals in which the connecting bridge had been broken, and in those animals in which the section was shown to be complete the dissociation was full and permanent.

That the impulse is conveyed from auricle to ventricle through the medium of the *A-V* bundle and through this channel only, is now an established and generally accepted conclusion. Compression or section of the bundle in its course from the node to its division is adopted in a number of laboratories as a preliminary procedure in experiments in which functional

dissociation of the two heart chambers is desired. In my own laboratory and for this purpose I use a clamp devised in conjunction with some observations made for me by Dr. Meakins (*547*). The blades of this clamp (Fig. 40) are introduced respectively into the right ventricle through the auricular appendix and through the aortic valves into the left ventricle *via* the carotid artery.

Fig. 40. (*Heart, 1913-14, V, 282, Fig. 1.*) ($\times \frac{1}{2}$.) A clamp devised for use in laboratory experiments, in which *A-V* heart-block is desired.

The curves of the clamp are suitably moulded so that the jaws of the instrument meet and squeeze the upper part of the ventricular septum, when the instrument lies in place. The clamp is easy to apply and heart-block of different grades may be obtained if graduated pressure is exerted.

For a description of heart-block itself the reader is referred to a later chapter.

Curves resulting from lesions of the chief divisions of the bundle in the dog.

When the impulse transmitted from auricle to ventricle has passed through the *A-V* bundle, it flows along two channels, namely, the right and left divisions of that bundle. That such is the case is suggested by the anatomical connections of these structures; it has been shown quite definitely by experiment, for Eppinger and Rothberger (*137*) found that separate transection of both divisions yields complete dissociation, as does a lesion of the main stem. The next reason for the conclusion is the remarkable change developing in the shape of the electrocardiogram when one or other division of the bundle is broken; for this change in the curve represents a profoundly altered distribution of the excitation wave, the impulse being conveyed to the opposite (contralateral) ventricle only. The excitation wave of the affected (homolateral) ventricle is started, not by an impulse transmitted through the proper channel—for this has been injured—but by spread through the musculature which unites the two chambers. It will be convenient completely to prove this statement in a subsequent

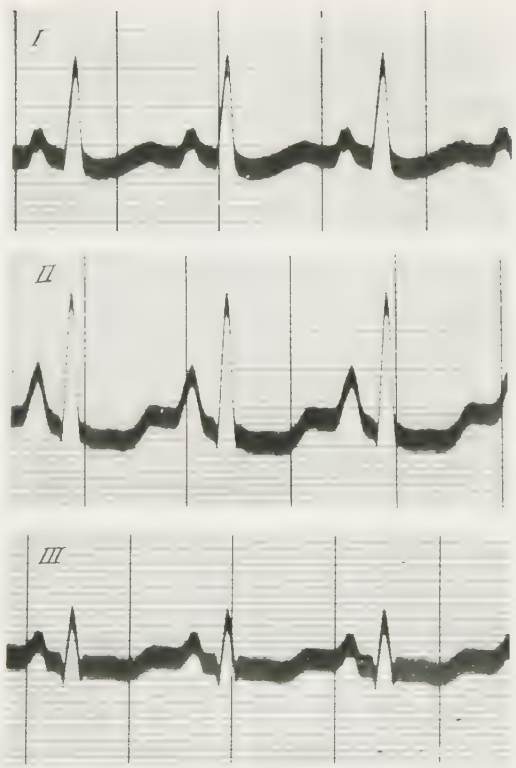


Fig. 41.

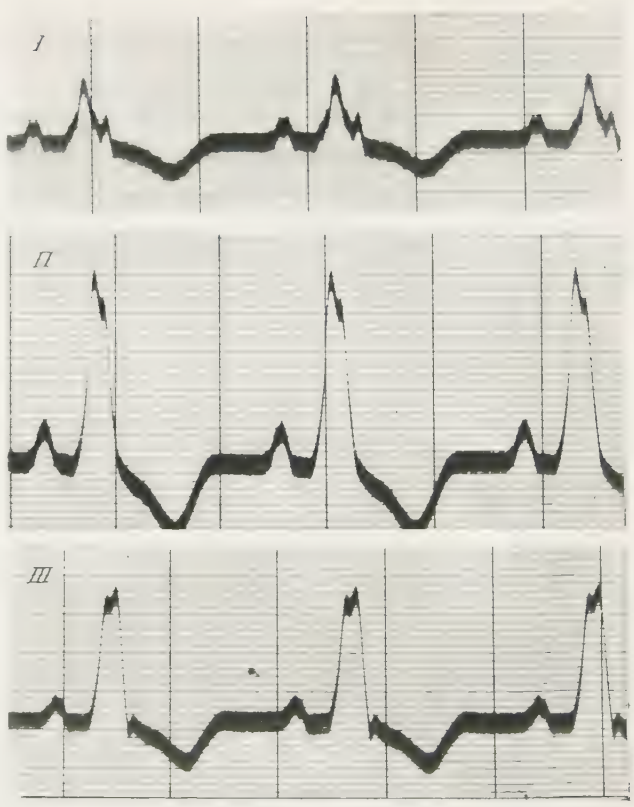


Fig. 42.

Fig. 41 and 42. (*Phil. Trans., 1916, B., CCVII, 243, Part III, Fig. 19 and 20.*)

Fig. 41. Electrocardiograms from leads *I*, *II* and *III*: showing the natural curves of a dog.
Fig. 42. Curves from the same animal and the same leads after cutting the left division of the *A-V* bundle Time in fifths of a second.

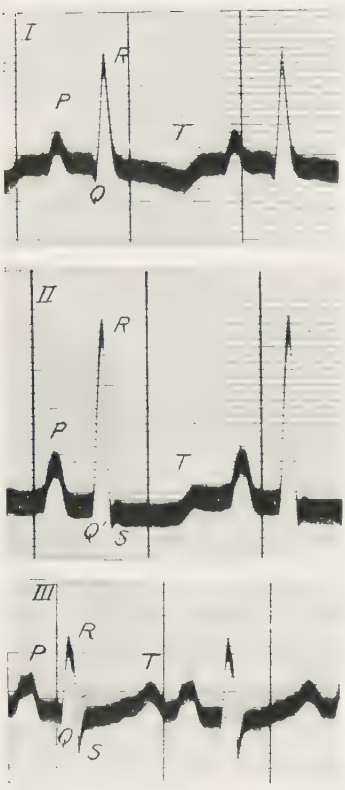


Fig. 43.

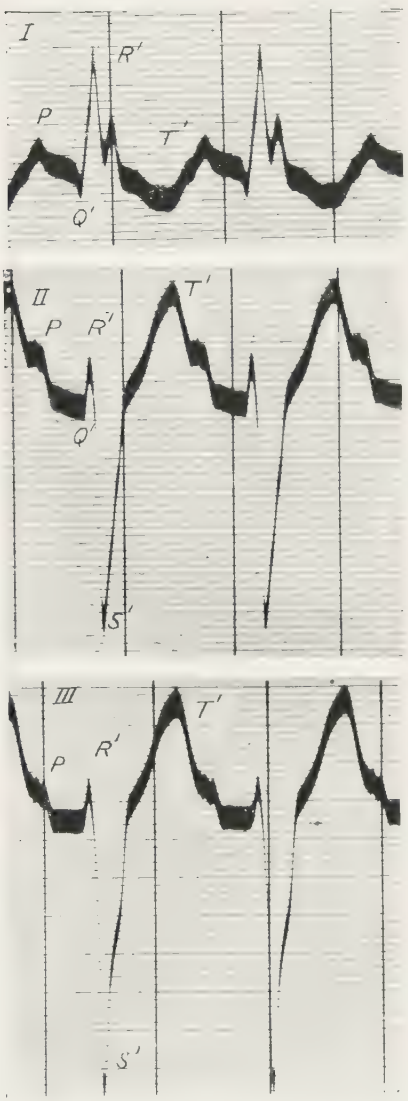


Fig. 44.

Fig. 43 and 44. (*Phil. Trans., 1916, B., CCVII, 243, Part III, Fig. 15 and 16.*)

Fig. 43 Electrocardiograms from leads *I*, *II* and *III*; showing the natural curves of a dog

Fig. 44. Curves from the same animal and same leads after cutting the right division of the *A-V* bundle. Time in fifths of a second.

chapter, and at present to be content with demonstrating that a lesion of either division of the bundle is definitely associated with a characteristic type of electrocardiogram, describing this in some detail.

Rothberger, working with Eppinger, was the first to describe electrocardiograms obtained from an axial lead in dogs subsequent to section of the separate divisions of the bundle, and in a second communication with Winterberg (*671*) the same worker amplified his account of the curves. Recently I have undertaken a series of experiments upon the same animal (*475*), confirming their chief conclusions, and I am in a position to describe the effects of the lesions in more detail.

A cut which traverses a division of the bundle is responsible for curves which, though open to certain variations, yet present characteristic general features. These features may be studied in Fig. 42 and 44. The curves in leads *II* and *III* are of exaggerated amplitude as compared to the normal curves, and in all leads the initial phases (R', S') are of considerable duration. The final deflection (T') is opposite in direction to the chief initial deflection (R' or S', as the case may be). These statements apply equally to lesions of either division.

In lesions of the *left division* (Fig.42), the chief deflections of leads *II* and *III* are R' and T', the former upwardly, the latter downwardly directed, forming as a whole a broadly diphasic effect. In lead *I* the deflections are generally of lesser amplitude, but similar in their directions.

In lesions of the *right division* (Fig. 44), the chief deflections of leads *II* and *III* are S' and T', the former downwardly, the latter upwardly directed, and forming as a whole a broadly diphasic effect, which is preceded by a dip, Q'. In lead *I* the first deflection may be upward (R'), and is then followed by a downwardly directed T'. In other, and perhaps more numerous instances, the deflections are small and directed as in leads *II* and *III*.

The curves contrast strikingly with the natural electrocardiograms in the same animals (Fig. 41 and 43), and are at once recognisable. They are always developed and are permanent when the corresponding bundle division is cut across ; they do not develop in these experiments unless (1) the section is complete, or unless (2) the section being partial, the remaining strands have been damaged by direct pressure or by that of the subendocardial effusion of blood ; in the last circumstances the abnormal curves are not persistent. In brief, a persistent change of the type described is always witnessed, and witnessed only when the track to one or other ventricle is rendered permanently non-conducting.

The curves are described at this stage because in studying the distribution of the excitation wave in the ventricle it is necessary that they should be understood. The clinical significance of the experiments will be discussed further in a later chapter. At this point I would utter the warning that the curves just described are those recorded from *dogs* ;

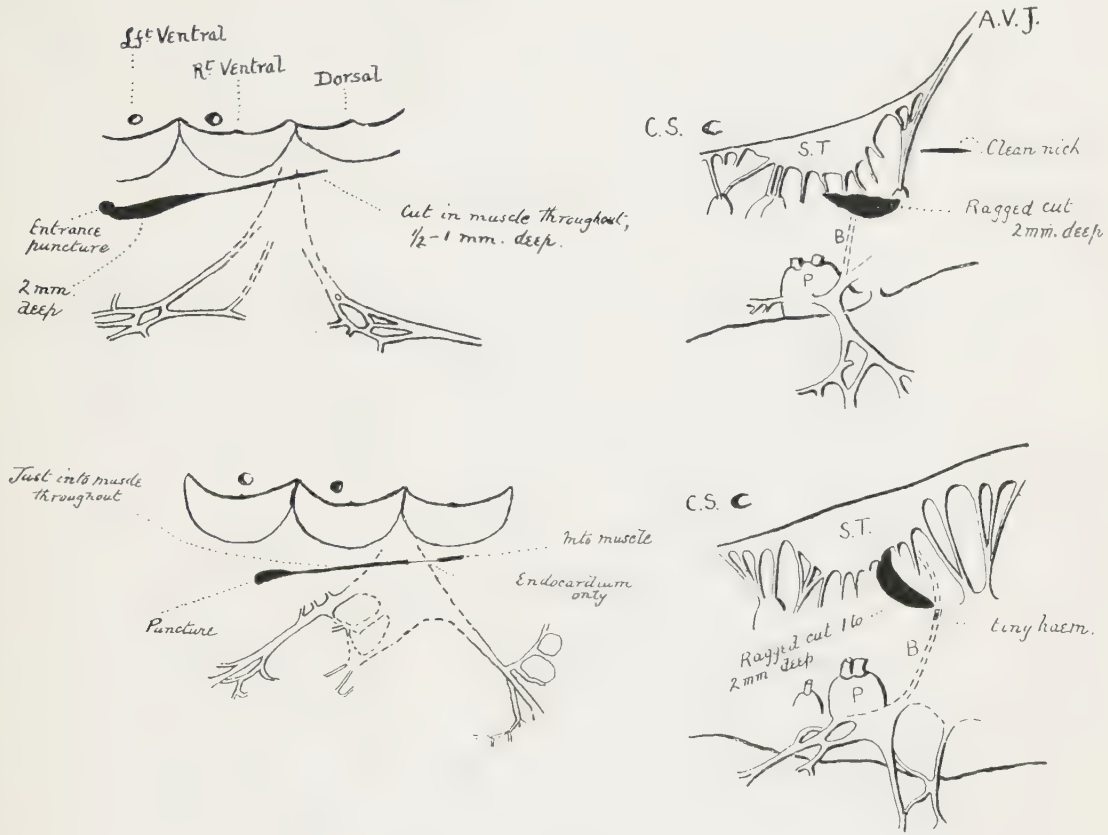


Fig. 45. (*Phil. Trans., 1916, B., CCVII, 255 and 266, Fig. 3 and 7.*) Drawings to scale of four experimental lesions in the hearts of dogs. The top left-hand figure shows the septum beneath the aortic cusps and the course of the left division of the bundle relative to an incision which breaks it completely near its origin; this lesion produced a permanent change in the electrocardiogram of the type shown in Fig. 42. The bottom left-hand figure is an example of a similar experiment in which the incision has not completely divided the conducting tissue, at one point the endocardium alone has been cut; the electrocardiogram changed to the type shown in Fig. 42, but only temporarily. The right-hand figures represent the right side of the ventricular septum in two further experiments: *C.S.* = coronary sinus, *A.V.J.* = auriculo-ventricular junction, *S.T.* = tricuspid valve, *P* = chief papillary muscle, *B* = right division, pursuing its course upon the septum. In the upper figure the incision has broken the conducting tract; the electrocardiogram changed to the type shown in Fig. 44. In the lower figure the incision is just clear of the tract, but has produced a tiny hæmorrhage into its sheath; the electrocardiogram changed to the type shown in Fig. 44, but only for a short while, and then returned to the normal form.

in certain broad features the corresponding human curves resemble them ; but the human curves, especially those which correspond to lesions of the left division, differ greatly from them in detail.

Note. For recent observations on A-V conduction in the lower vertebrates the following papers should be consulted (*175*, *415*, *416*, *417*, *531*, *533* and *571*).

CHAPTER VII.

SPREAD OF THE EXCITATION WAVE IN AURICLE AND VENTRICLE.

In the auricle.

IT has been shown that the natural pacemaker of the heart has its seat in the upper part of the *sulcus terminalis*; the heart beat is initiated in, and the excitation wave spreads from, the sino-auricular node. The progress of the excitation wave through the auricle may now be studied. It is examined in one of two ways (*483*).

If outlying leads are used (Fig. 46), the contacts being arranged radially to the *S-A* node, then the direction of the first prominent or "intrinsic"

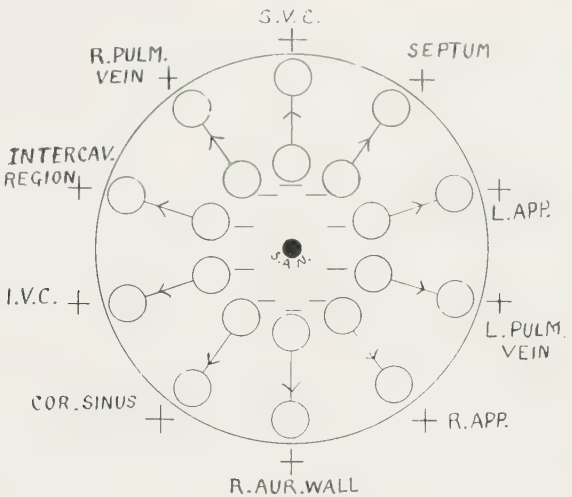


Fig. 46. A diagram to illustrate a system of "outlying" leads. Paired contacts are arranged radially to the node and in contact with different parts of the auricle. The direction of the intrinsic deflection, in these circumstances, demonstrates that the proximal contact (nearest to the *S-A* node) always receives the excitation wave before the distal contact.

deflection of the electrogram will teach us which of the two contacts of a given lead is first influenced by the excitatory process as it travels. A general statement may be made in regard to the whole auricular superficies, including the muscular sleeves which embrace the inlets of the veins. Wherever

the double contacts are placed, that contact which lies nearest to the *S-A* node shows relative negativity while the intrinsic deflection is inscribed. From the directions of the intrinsic deflections alone we may conclude that the excitation wave spreads radially in every direction from this node; that it runs down the tænia, that it courses from the base to the tip of the right appendix, that it moves along the intra-auricular muscle strand to the tip of the left appendix, that its progress is down the septum, that it runs in the muscle sleeves of cava, coronary and pulmonary veins against the blood stream.

These conclusions are fully established by the second method of investigation, namely, by timing the onsets of intrinsic deflections corresponding to different contact areas, against a standard deflection in an axial electrocardiogram. If a series of contacts is laid in line along the *tænia terminalis* as in Fig. 47, and the times at which the excitation wave

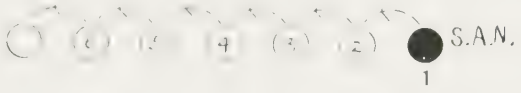


Fig. 47. A diagram to illustrate leads from contacts in series. The head of the *S-A* node lies at the one end of the series, and the time at which the excitation wave appears at each contact of the series is estimated by leading from each succeeding pair. It is found that the time increases steadily, and, when the contacts are equidistant, by equal increments as the leads recede from the *S-A* node.

arrives at contacts 2 to 6 are estimated, relative to its arrival at contact 1, these readings are found to increase in a regular order. The excitation wave moves at a uniform rate from *S-A* node to the end of the tænia.

READINGS FOR CONTACTS ARRANGED SERIALLY AND IN THE LINE OF THE *S-A* NODE ON THE TÆNIA AND CAVÆ.

Tænia contacts 8 mm. apart.	Superior caval contacts 5 mm. apart.	Inferior caval contacts* 5 mm. apart.
·0000	·0131	·0201
·0083	·0194	·0238
·0159	·0265	·0287
·0200	·0325	·0343
·0251	·0357	·0394
		·0447
		·0519

Readings for contacts *a* to *g*, in Fig. 48

Fig. 48. (*Phil. Trans., 1914, B., CCV, 375, Fig. 14.*) A diagram of a dog's auricle to scale, showing two series of contacts on the inferior cava. The muscle bands are shaded. *T.T* = tænia terminalis. The curves obtained from each pair of contacts, *a*-*b*, *b*-*c*, etc., to *h*-*i*, are charted relative to the same standard time instant in Fig. 49.

A similar proof is forthcoming that the excitation wave moves up the superior cava and down the inferior cava. Thus in Fig. 48, the contacts *a* to *g*, lying on the tænia and inferior cava, gave the readings shown in the last column of the above table.

The corresponding electrograms are illustrated in Fig. 49; all these curves are plotted relative to the same standard, namely, the time reading of the excitation wave at its onset in the *S*-*A* node; the gradual recession of the intrinsic deflection as the lead is moved away from the node is clearly shown. It is noteworthy that the intrinsic deflection is always inscribed when one contact lies over auricular muscle, but when the edge of this muscle is passed (as in lead *h*-*i*, Fig. 48) the intrinsic deflection disappears (Fig. 49).

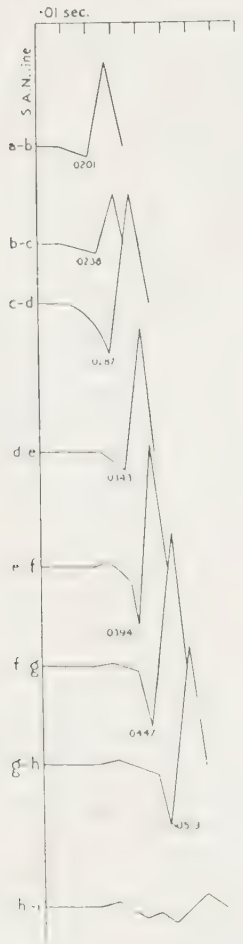


Fig. 49. A series of electrograms charted in relation to the *S.A.N.* line (the line representing the onset of the excitation wave in the auricle). They were taken from the corresponding leads *a-b*, etc., depicted in Fig. 48.

Investigating the auricle in this fashion, and knowing the distances between given contacts and the *S-A* node, we may estimate the rates of conduction to all parts of the auricle.

TIMES AND RATES OF TRANSMISSION OF THE EXCITATION WAVE TO VARIOUS PARTS OF THE AURICLE.

Region.	Average distance in mm.	Average transmission, time in seconds.	Average transmission, rate in mm. per sec.	Number of observations.
Intercaval region	15·2	·0139	1232	18
Intra-aur. band	12·9	·0126	1252	6
S.V. cava	8·2	·0136	588	11
Septum (mid & low)	31·5	·0305	1059	11
Rt. appendix	28·0	·0314	955	11
Rt. auricle	16·0	·0206	859	7
Rt. pulm. vein	24·0	·0254	1121	4
I.V. cava	31·5	·0325	998	18
Coronary sinus	43·9	·0412	1096	5
Left pulm. vein	45·2	·0412	1118	5
Left appendix	44·6	·0446	996	7

Average heart rate 158·4.

These calculated rates, are, with minor exceptions, wonderfully uniform, such differences as occur being explained by error of measurement and by the arrangement of the muscle bands. The chief variant is the superior cava, where, apparently on account of the obliquity of its fibres, the rate is notably slower.

There is no evidence of slow conduction from the node itself to adjacent muscle fibres, neither can I accept the observations of Eyster and Meek (*161, 163*), as showing that there is more rapid conduction from *S-A* node to *A-V* node.* The wave spreads between the two nodes through the muscle of the septum, as it does through other auricular muscle bands, at a rate approaching 1,000 mm. per second.

* If we are to accept the findings of these workers, it must be allowed that the spread of the excitation wave in the auricle varies fundamentally in its course through the auricles of different dogs. Such variations are opposed in general by physiological experience, and in particular by the considerable uniformity of the *P* summit in lead *II* in different animals. Eyster and Meek's conclusion is based chiefly upon study of curves obtained from paired contacts on the auricle. A contact is placed on each of two points under observation and the two contacts are led to the galvanometer; the point first showing relative negativity is ascertained. This method is fallacious, as I have pointed out (*483*), if it takes no account of extrinsic effects; in practice the distinction between extrinsic and intrinsic effects is often difficult or impossible, if the lead is not in the line taken by the excitation wave.

The spread of the wave may be likened to the spread of fluid poured upon a flat surface (*474*), its edge advancing in an ever widening circle until the whole surface is involved (Fig. 50).

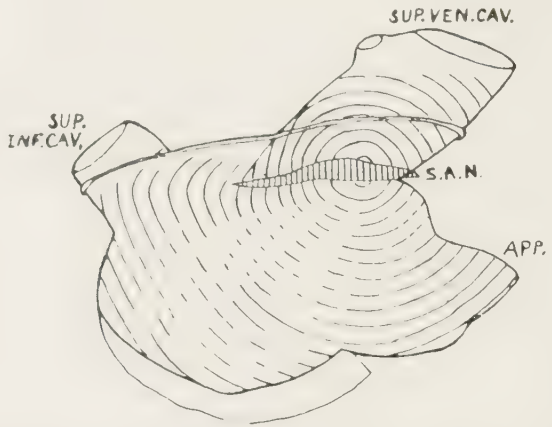


Fig. 50. A diagram to illustrate the manner in which the excitation wave spreads over the surface of the right auricle. The spread is from the upper part of the node and follows the chief muscle bands at almost uniform rates.

The arrangement of the auricular muscle guides this mode of spread. The chief bands run from the region of the *S-A* node. This node is placed in the most advantageous position for quick distribution, the muscle fibres run from its neighbourhood in distinct bands (see Fig. 2, page 2); the tænia runs from the top to the bottom of the sulcus; the strong intra-auricular band runs from the top of the sulcus, behind the aorta to the tip of the left appendix; the chief muscles of the right appendix radiate from the tænia; other fibres run from the sulcus down the septum.

To sum up: the excitation wave, originating in the *S-A* node, spreads at once and at rates ranging around 1,000 mm. per second along the chief auricular muscle bands and these radiate from the neighbourhood of the node. It spreads as fluid does when poured on a flat surface, involving an ever increasing area, and finally progresses against the blood stream at the mouths of the veins to end upon these where the muscular sleeves end. It reaches the *A-V* node by spreading through ordinary auricular muscle, and passes into the *A-V* bundle. Its course through this bundle was proved in the last chapter.

In the ventricle

The problem of spread in the ventricle is more difficult; for the events succeed each other with greater rapidity. It has been studied in the first instance by examining the surface distribution of the wave (*486*), and for this purpose a single contact is placed upon the epicardium, while a second

0241
0251
0231
0151
0178
0167
0073
0082
0213
0146
0150
0187
0190

Fig. 51. (*Phil. Trans., 1915, B., CCVI, 181, Fig. 21.*) A projected drawing ($\times \frac{7}{10}$) of the interior of a dog's right ventricle, showing the relations of the large papillary muscle and free arborisation of the right bundle division to the surface. The overlying tracings are projections of the superficial muscle fibres and of the surface of the heart with the contacts and readings of the experiment.

rests upon the body wall. The intrinsic deflection, consequent upon the arrival of the excitatory process beneath the epicardial contact, is always upright in the corresponding curves and may be clearly recognised and timed if this method is constantly employed.* The surface distribution in the dog may be exemplified by Fig. 51.

If a series of readings from contacts overlying a superficial band of muscle fibres is studied, for example the muscle band which sweeps from the conus across the upper part of the interventricular groove and around the left border of the heart to the apex, it is at once evident that the wave of excitation does not follow the band. Fig. 51 serves as an illustration ; in this instance the readings ·0241, ·0231, ·0198, ·0150, ·0146, ·0187 and ·0196 sec.,† are found along the muscle band in question. It is activated almost

Fig. 52. (*Phil. Trans., 1914, B., CCVI, 196, Figs. 6 and 9.*) ($\times \frac{2}{3}$.) Surface distribution in two dogs over right and left ventricles, seen from the sides. *L.Vo* = Vortex of left ventricle.

simultaneously throughout its whole length. This and other observations clearly show that the arrangement of the muscle bands does not materially influence the spread, as is the case in the auricle.

* Erfmann (*141*) has used Clement's differential electrodes (*51*) (*i.e.*, closely paired contacts on the ventricle), but the results obtained by this method are open to objection in that while intrinsic and extrinsic deflections are recorded, these often cannot be distinguished in leading from the ventricle, *because the direction of the intrinsic deflection is not actually known beforehand.* In practice the use of differential electrodes has not proved satisfactory when exploring the ventricle.

† These readings are the time intervals between the beginning of *R* in lead *II* and the arrival of the excitation wave at the corresponding contact points.

If readings are taken from the whole front surface of the heart, then the region of the right ventricle that lies parallel to the ventral attachment of the free wall to the septum is always found to be activated first ; but this area, in which the excitation wave appears early is of considerable extent ; many points are activated almost simultaneously and, if the underlying structures are examined, it will be found that the area corresponds to the free arborisation of the right bundle division, an arborisation which is very prominent in the dog (Fig.51). Now the rest of the right ventricular surface (front and back, see Fig. 52) is activated later and in a constantly increasing degree as we pass towards the base of the heart. Although this order of the readings is constant, yet the time differences between them are so small that the order cannot be explained by direct surface spread at the transmission rate ascertained for ventricular muscle. (This, as we shall see, is about 400 mm. per sec..)

In the case of the whole auricle, the progress of the wave from start to finish occupies some four or five hundredths of a second ; in the ventricle, despite the larger size of this chamber and despite the slower conduction rate of its muscle, the surface is supplied in three-hundredths of a second or less. The order over the left ventricle is quite as definite (Fig. 52) but it need not be described in detail. Suffice it to say, that the vortex (*L.Vo*) is the first surface point activated, while the central and basal parts are excited later, and that over a large part of the central region the wave appears at a number of points almost simultaneously.

No system of spread from point to point of the muscle fibres can be imagined to explain this distribution. We are compelled to assume that different parts of the surface are activated along distinct channels. These channels, as can be shown, are constituted by the arborisation and network of the bundle divisions, and the excitation wave spreads in the ventricle from within outwards.

The Purkinje pathway.—In a previous chapter, changes in the type of the electrocardiogram have been stated to accompany lesions of the chief branches into which the bundle divides. The reason for this change, namely, altered distribution of the excitation wave, can now receive proof. If readings are taken from a series of contact points on right and left ventricle (Fig. 53) before (upper readings) and after (lower readings) the termination of the right branch (*R.B.B.*) has been divided, it is found that the readings over the left ventricle remain unaltered.* But over the front of the right ventricle the change is profound and the magnitude and order of the readings is such that the right ventricle is clearly shown to be activated by spread from the left ventricle. Similar, but even more profound changes are seen when the left branch is divided at or near its point of origin.

* Except for a very small and constant difference, which is attributable to alteration in the shape of the standard curve of measurement.

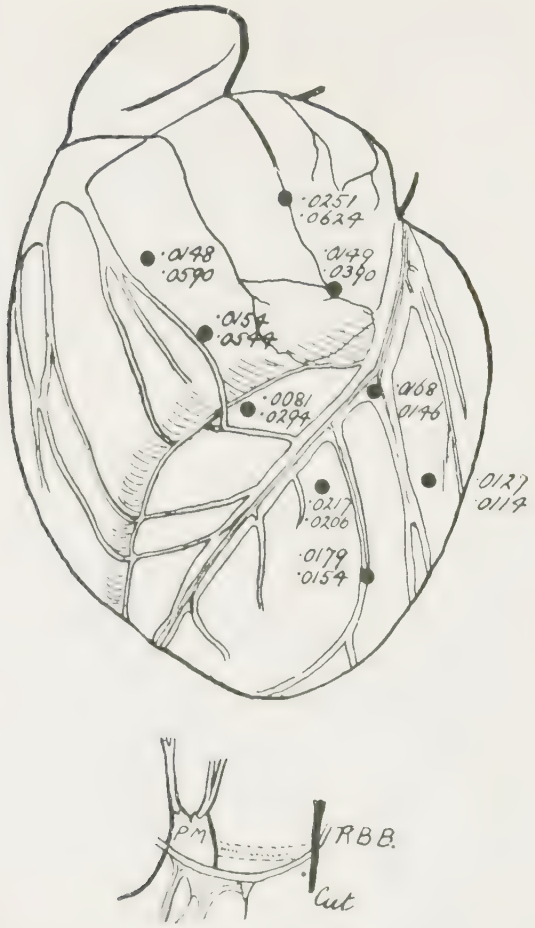


Fig. 53. (*Phil. Trans., 1914, B., CCVI, 200, Fig. 12.*) Natural size projection of ventral surface of a dog's heart, showing readings for a series of contacts before (upper reading) or after (lower reading) cutting the right division of the bundle. The line of union of the two ventricles is marked by the chief blood vessel. The cut and its relation to the branch (*R.B.B.*) and papillary muscle (*P.M.*) is shown below.

The main divisions of the bundle are thus proved to be concerned in distributing.* It can also be shown that the network of Purkinje is concerned ; for if a reading is taken from the surface of the conus, a transverse cut or even a scratch, placed on the apical side of the contact, conspicuously delays the reading, providing the cut or scratch is upon the endocardial lining of the heart ; a similar cut in the epicardial surface, even though it penetrates

* The branches are capable of conducting in either direction (*486*) though normally they conduct only in one.

the muscle deeply, is without effect; the spread takes place along the endocardial surface. Furthermore, if four deep cuts are made on the surface, so as to surround a surface contact, then from this contact the readings before and after the interference are the same. No experiment points more definitely to the progress of the excitation wave through the muscle from within outwards; none shows more distinctly that the wave is independent of the direction of the muscle bands.

Conduction rates.—If an artificial wave is promoted by stimulating the surface of the heart in line with two contacts (Fig. 54), the time interval

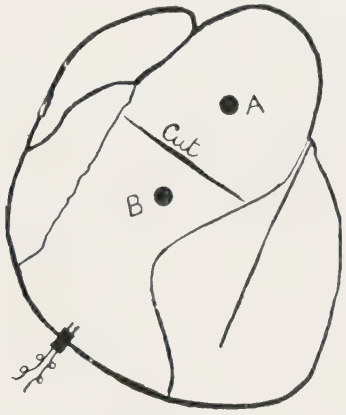


Fig. 54. (*Phil. Trans., 1914, B., CCVI, 201, Fig. 14.*) A diagram illustrating an experiment in which it is shown that the conduction interval between two contacts is uninfluenced by cutting the superficial muscle fibre lying between them. Each muscle contact is paired with its own chest wall contact, and the records are taken simultaneously. The stimulating electrode is placed in line with the two heart contacts.

between the activation of the muscle under the two contacts can be accurately determined, and a rate of transmission can be calculated. For different parts of the ventricular surface this rate varies. It is highest and approaches or surpasses 2,000 mm. per second where the muscle wall is thinnest; is lowest and approaches 400 mm. per second where the muscle is thickest. The reason for this variation is that the rate of conduction in muscle proper is slow, while in Purkinje substance it is very rapid. If the wall is stimulated where it is thin, the artificial wave penetrates the whole muscle thickness and is conveyed along the Purkinje network, from which it spreads outwards through the muscle again to reach the contacts; if, as in the left ventricle, the wall is thick, the wave may be conducted across the superficial contacts before it travels to the lining of the heart and out again. These conclusions follow from experiment. The border of the right ventricle is stimulated (Fig.54) and an artificial excitation wave is propagated

from B to A ; the times of arrival at the two points is ascertained. A deep cut in the muscle between the contacts does not affect these readings ; a shallow cut or scratch on the endocardial surface at once delays the arrival of B. Clearly the wave is carried along the endocardial lining over the greater part of its course.

If contacts are placed on the pericardial surface (P) and on the endocardial lining (E) of the wall (Fig. 55) and the surface is stimulated

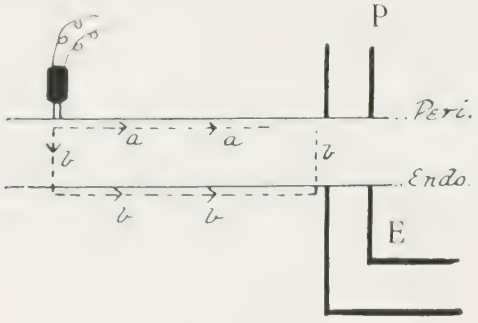


Fig. 55. (*Phil. Trans., 1914, B., CCVI, 208, Fig. 15.*) A diagram showing the alternative paths (*a-a, b-b*) which an artificially induced excitation wave may take in the heart wall.

at some distance from them, the excitation wave is found *to reach the internal contact first* and, if the stimulating electrodes are far enough removed, the interval between the readings at the two contacts is *precisely the same as for the natural heart beat* (natural readings).

In brief, the excitation wave reaches P by the path *b-b*, for that path is the quicker on account of the high rate of conduction in the lining. But if the stimulating electrodes are nearer to the contacts, the wave may reach P along the path *a-a*. If the thickness of the muscle wall is known and the distance from the point of stimulation is also known, then, in an experiment in which the natural readings at the two contacts are just maintained, the relative rates of conduction in Purkinje substance and muscle can be ascertained. The rate is at least five times more rapid in the network than in the muscle. The experiment is arranged so that the excitation wave reaches P along the two paths *a-a* and *b-b* simultaneously ; the length of muscle traversed along *a-a* and the length of muscle and network traversed along *b-b* is ascertained and from these data the conduction rates are calculated. When the results of experiments of this and other kinds are considered, it is calculated that in muscle the rate of conduction is in round figures 500 mm. per second ; in *straight* (as opposed to the usual wavy) Purkinje strands it is approximately 5,000 mm. per second.

Further arguments.—When the heart beats naturally, readings from the lining of the heart are always earlier than readings from the surface at corresponding points, and the difference between such readings is controlled by the thickness of the intervening muscle. Readings from the lining of the conus are later than readings from other parts of the lining of the right ventricle ; and this is so because the Purkinje path to the conus is the longest.

Thus the distribution at any point on the surface of the ventricle is controlled by two factors, the length of the Purkinje path, and the thickness of the muscular wall ; if these data are ascertained for a number of surface points, and calculations are made on the basis of estimated transmission rates in the two classes of conducting tissue, then these calculated readings are found to correspond in a surprisingly exact manner with actual readings from the surface. The chief controlling factor is the muscle thickness, and it is partly for this reason that the attached border of the right ventricle is activated early, for here the muscle is thin ; it is wholly for this reason that the vortex of the left ventricle is activated early, for here the muscle is often extremely thin.

Certain time-relations between the spread of the excitation wave in the heart and the deflections of the axial electrocardiograms are of importance. Activity begins in the auricle at an average time interval of 0·01 of a second before the beginning of the deflection *P*. Activity in the ventricle begins approximately 0·005 of a second before the beginning of *Q* in an axial lead. The galvanometric string, arranged at tensions such as it is customary to employ, fails to record the progress of the excitation wave in a limb lead, until a considerable mass of muscle is involved. A comparison of the surface and lining readings shows clearly that the whole period of the initial phases, *Q*, *R* and *S*, is occupied by the spread and development of the excitatory process in the ventricle. *The total duration of these deflections corresponds to the activation of the muscle** and it constitutes an approximate measure of the duration of the spread.

A final word completes this chapter. The conduction rates in the heart muscle increase as we pass from ventricular muscle to auricular muscle and from these to Purkinje tissue. The glycogen content of these tissues and the breadth of the fibre increases in the same order. The *A-V* node has the finest fibres and the glycogen content is poor (*569*) ; there is much evidence in proof of its having the lowest conducting power. Thus, there is a suggestive relation between the power to conduct and structural and chemical constitution.

Distribution in the auricle is expedited by the central position of the pacemaker, by the arrangement of the muscle and the relatively high rate of muscle conduction (*i.e.*, 1,000 mm. per second). The muscle of the ventricle conducts slowly (*i.e.*, 500 mm. per second) because its function of distributing

* A conclusion which applies not only to the dog's heart, but to the amphibian, reptilian and avian heart (*475*).

is a minor one; but this, the driving chamber, is provided with a special system of distribution, clearly arranged to provoke almost simultaneous contraction of all parts of the walls; these special fibres are endowed with conduction powers of the highest order (5,000 mm. per second).*

NOTE.—In this chapter it has been possible only to summarise recent observations; for further details of the work the original papers should be consulted. It has also been considered undesirable to expand the chapter by including a description and criticism of many earlier attempts to trace the course of the wave of excitation and contraction. A number of the earlier writers sought to unravel the excitation wave in the ventricle by analysing the curves obtained from paired contacts resting on the ventricle; the last exponent of this method was Gotch (*230, 231*). The method is insufficient and Gotch was led astray, as were his predecessors, by treating the whole ventricle as a simple strip of muscle. These workers were either unacquainted with or failed to grasp the high significance of the Purkinje conducting system. This historical aspect is dealt with in a recent paper (*475*).

It is also unnecessary to describe the relatively crude attempts to determine the precedence of contraction in the two auricles or two ventricles by mechanical devices (*677*), or of different portions of one ventricle by the same means (*139, 281*). There are parts of the right auricle which contract before parts of the left auricle, there are parts of the left which contract before parts of the right; the same statements apply to the ventricles. Mechanical methods of recording, even as they are now developed, are impotent to solve such questions. The order of contraction may be presumed to be the order of activation, and that is described in the present chapter.

NOTE.—For recent observations on the spread of the excitation wave in the ventricle of the lower vertebrates the following papers may be consulted (*51* and *475*).

* Some direct measurements of the rate of conduction in Purkinge strands have been obtained by Erlanger (*146*). The rate as estimated by him is 750 mm. per second. The observations were undertaken in the perfused heart of the calf, and therefore in all probability underestimate the Purkinje conduction rate very considerably.

The same writer has shown that Purkinje fibres are excitable to artificial stimulation, but whether they contract or not is still unknown. Their striation certainly suggests this power, as does also the elaborate protection by sheaths which they enjoy in the ungulates.

Chapter VIII.

THE MEANING OF CERTAIN VENTRICULAR DEFLECTIONS.

Q, *R*, *S*, THE INITIAL DEFLECTIONS.

The duality of the normal electrocardiogram.

In the last chapter it was shown that the excitation wave in spreading throughout the ventricle follows the branches of the *A-V* bundle and the arborisation of Purkinje; the wave courses from the endocardial to the pericardial surface of the musculature. During the stage of invasion it gives rise to the initial deflections of the electrocardiogram. In the first part of the present chapter, the constitution of these deflections will be described in so far as it is at present understood. It may be reiterated that we have proof that *Q*, *R* and *S* correspond to the stage of invasion or spread, for the time interval covered by these deflections corresponds with that covered by direct readings from the ventricle. If the diagram (Fig. 29, page 54) may be used for comparison,* these deflections correspond to the *first phase* of the curve there illustrated.

Inasmuch as the excitatory process passes to the ventricle by two distinct channels, formed by the right and left divisions of the bundle, and since no anatomical union is known to exist between the arborisations of the two sides, it might be assumed that the spread to a given ventricle is a distinct process and is confined to that ventricle; that is to state that the excitation wave does not cross the interventricular groove during the natural heart beat, but that the right and left waves meet somewhere in the septum. This assumption is supported by two considerations. First, it is supported by the actual readings from the surface of ventricles, for these show an abrupt change of order in the region where the two ventricles meet (*486*). Secondly, it is supported by the effects of branch lesions already described. When one division is cut, the distribution to the contralateral ventricle is unaffected right up to the septum, for the excitation wave spreads in normal fashion through the uninjured division; but the spread through the homolateral ventricle is completely altered and now proceeds across the septum from the ventricle whose supply is uninjured.

* Strictly speaking, I do not think this comparison is quite justified, in that *Q*, *R* and *S* are written in an indirect lead and the diphasic curve of Fig. 29 is obtained by a direct lead. Actually, however, a very similar diphasic curve is yielded by leading indirectly from a simple strip of muscle.

This being the case, we are justified in supposing that the natural ventricular electrocardiogram is in reality a composite picture, depicting the superimposed effects of the separate ventricular activities.*

This assumption has recently been proved correct by means of experiments devised to that end (*475*). When a single division of the bundle is transected, the excitation wave passes naturally to the opposite ventricle; it courses through it in a normal fashion but yields a highly abnormal curve, for the ventricle is then activated in the absence of a balancing activity in the other chamber. But the curve may be termed *normal* in the sense that it represents the electrical events so far as the activation of this normally excited ventricle is concerned. This statement clearly applies to that portion of the curve which represents spread in the ventricle directly supplied from the auricle; it does not apply to that portion which represents cross spread (abnormal spread) in the other ventricle. It applies, as has been shown, to the preliminary phases, or the sharp deflections, of the electrocardiogram. If the preliminary deflections are produced by spread confined to the right ventricle (as when the left division is cut), I term the curve a *dextrocardiogram*:† if by spread confined to the left ventricle (as when the right division is cut), a *levocardiogram*.† Now it is possible to obtain both the dextrocardiogram and levocardiogram of the same animal in experiment by alternately squeezing the left and right divisions of the bundle, and temporarily throwing them out of action as conducting structures. It is also possible, by modifying a method of mensuration described in a previous chapter, to chart these curves, so that corresponding time phases lie vertically the one above the other (Fig. 56).

When such a chart is accurately constructed, we have before us a complete and accurate statement of the electrical forces which combine to form the physiological electrocardiogram; and being complete and accurate, both in respect of voltage and time, the physiological curve may be constructed from them accurately and in detail by a simple mathematical process. The two curves are combined by algebraic addition. Thus, if on a given time line, the voltage in one curve is 10 and in the other curve —8, the corresponding voltage in the combined curve is 2. The chart (Fig. 56) shows the dextrocardiogram (Rt.) and levocardiogram (Lf.) of a dog as recorded electrocardiographically; it shows the calculated normal curve (*C*) and the actual normal curve (*N*) in the same animal. The calculated and actual curves are alike in shape, they are alike in their amplitudes and have similar time relations.

The proof is forthcoming, therefore, that the first portion of the normal electrocardiogram is a composite of two curves; it represents the summated effects (for other examples of summation, see page 158) of right and left

* An idea which has been mooted already as a hypothesis by Selenin (*696, 697*), Rothberger and Winterberg (*666*), and others.

† In my original paper (*475*), I spoke of dextrogram and levogram. The present terms are modified to suit Samojloff's terminology.

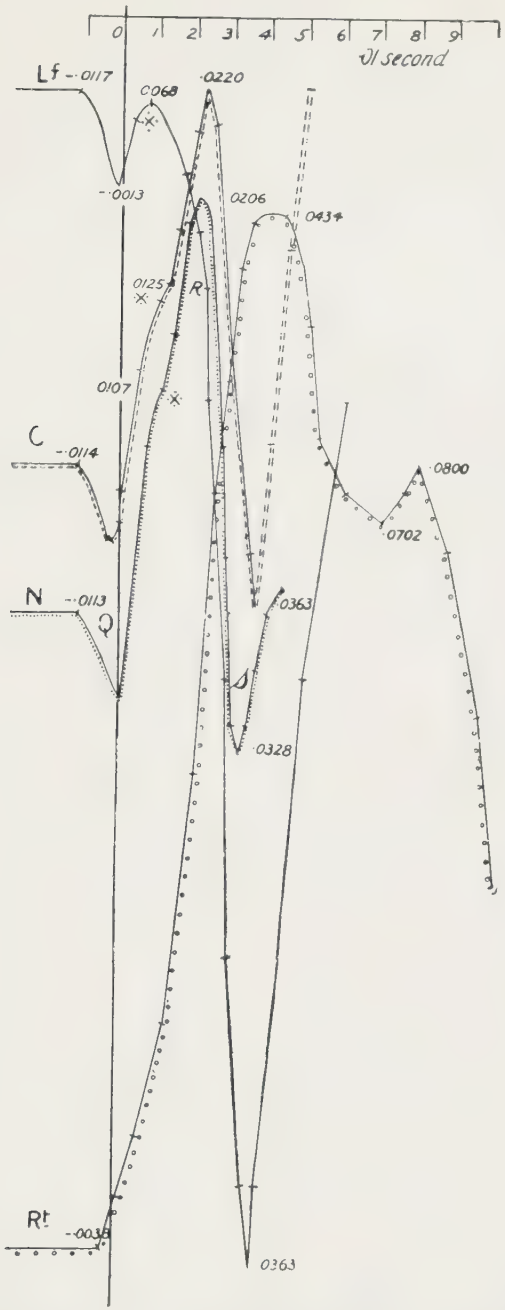


Fig. 56. (*Phil. Trans., 1916, B., CCVII, 251, Part III, Fig. 2.*) A chart of four curves. *Lf* is the levocardiogram, *C* is the calculated bicardiogram, *N* is the actual bicardiogram, and *Rt* is the dextrocardiogram. All the curves are plotted in relation to *R* of the natural bicardiogram, and this stands on the 0 line. The calculated bicardiogram (*C*) has been constructed by algebraic addition of the values found on corresponding vertical lines in dextrocardiogram and levocardiogram. It is an almost exact replica of the actual bicardiogram, both in respect of its time relations and in respect of the voltages represented by the several deflections. Note especially the notches marked by asterisks on the upstrokes of *R*. The calculated curve departs from the outline of the natural curve approximately some 0.0350 sec. after *R* begins, or approximately 0·0450 sec. after the first phase of the levocardiogram. Ordinates are on the scale of 7·5 cm. = 1 millivolt. Abscissæ, 1 cm. = 0·02 sec..

ventricles. Because it is a composite curve, consisting of the summated effects of right and left ventricle, I term it here the *bicardiogram*.*

The next step in the analysis of the normal electrocardiogram or *bicardiogram*, as it may be termed, consists in a separate analysis of levocardiogram and dextrocardiogram. To this end a different method is adopted. It is that of calculating the electrical axis.

Method of calculating the electrical axis.

First let it be stated that the electrical axis of the heart is constantly changing direction during the progress of systole and that our calculation is of the axis at a given instant in time.

To calculate the electrical axis† of the heart at a given instant in time, it is necessary to use more than one lead. It has become customary to use leads along the three sides of an imaginary triangle and the curves obtained depend upon the potentials developed at the angles of this triangle. As in moving, in the direction of the leads, along lead *I* and lead *III*, we arrive at the same point as in moving along lead *II*, Einthoven (*119*) has formulated the rule that $e^1 + e^3 = e^2$, where e^1, e^2, and e^3 represent the potential differences in the three leads. That the formula is a correct expression for electrocardiograms as we take them has been shown by actual observation.‡

In most electrocardiograms the general truth of this relation can be seen in comparing the values of the deflections in the three leads. To take an actual example : the lengths of the deflections in the three leads from a normal subject were as follows :—

	P	*Q*	*R*	*S*	*T*
Lead *I*	0·5	0·0	2·5	2·3	2·5
Lead *III*	1·0	1·3	10·0	1·0	1·0
Lead *II*	1·5	1·3	12·0	3·0	3·0

It will be seen at once that the values in lead *II*, approximate to the sum of the values in the remaining leads. But, as has been pointed out, crude additions of this kind are not always accurate since the incidence of a given summit is not always precisely the same in point of time in the three leads (*359*). If the exact time-relations of electrocardiograms from the three leads are ascertained one to another, they can be charted the one above the other so that

* It should be emphasised that the foregoing conclusions are applied solely to the electrocardiogram's initial phases, namely, to the deflections of the *Q*, *R*, *S* group.

† This is not to be confused with an anatomical axis ; no electrical axis is an exact guide to the latter, though movements of the electrical axis of a given phase of succeeding systoles may be read as indicating a similar movement of the anatomical axis (see page 127).

‡ The formula itself is necessarily true ; that it is found to apply accurately to electrocardiograms is but a demonstration that these curves are correct expressions of the electrical events.

corresponding time phases fall on the same vertical, and it is then shown that there is an absolute mathematical relation between the curves, providing that the curves are taken with a sufficiently care and with an accurate instrument. It follows, therefore, that if the values and times of the deflections in two leads are known, the curve of the remaining lead can be calculated mathematically with considerable accuracy.

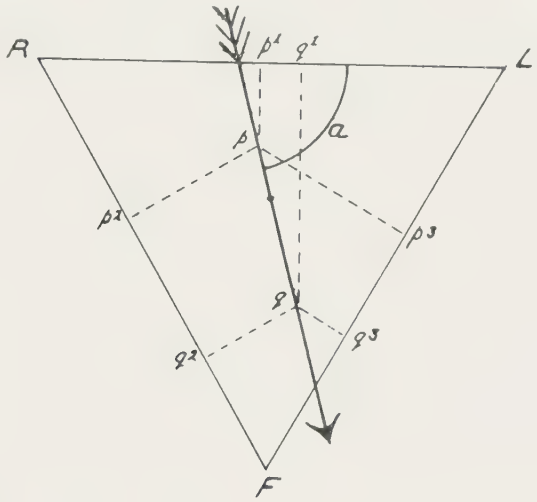


Fig. 57. (After Einthoven, Fahr and Waart. *Archiv. f. d. ges. Physiol., 1913, CL, 308, Fig. 22.*) A diagram of the axis of an electromotive force in the body (represented by the arrow) and its distribution along the sides of an equilateral triangle *R L F*; the angles of the triangle represent in an approximate fashion the points of contact in the usual leads adopted in electrocardiography. The line $p^2\ q^2$ is equal to the lines $p^1\ q^1$ and $p^3\ q^3$ taken together. The angle *a* which the arrow makes with the horizontal *R L*, is the angle of the axis referred to in the text.

The relative values in the several leads has been illustrated by Einthoven, who uses an equilateral triangle for the purpose; the sides of this triangle are taken to represent the three usual leads. While a particular summit of the electrocardiogram is being inscribed, the corresponding axis of potential is not necessarily that of either lead; but this axis can be calculated trigonometrically or geometrically if the potentials of two leads are known (Einthoven).

Trigonometrical method. (*Einthoven's formulæ (119).*)—An arrow is drawn through the mid-point of the equilateral triangle *R L F* (Fig. 57) and this arrow makes an optional angle *a* with the side *R L*. On this arrow, a line *p q* of optional length is taken. The projections of *p q* to the sides of the triangle are $p^1\ q^1$, $p^2\ q^2$ and $p^3\ q^3$. If *E* is the manifest potential difference*

* The potential difference indicated by a lead from contacts in the line of the electrical axis.

developed along the line of the electrical axis and e^1, e^2, e^3 are the potential differences as they are represented in the sides of the triangle, then the values of e^1, e^2 and e^3 are proportional to the lines $p^1 q^1$, $p^2 q^2$ and $p^3 q^3$ and

$$1. \quad e^1 = E \cos a$$
$$2. \quad e^2 = E \cos (a—60°)$$
$$3. \quad e^3 = E \cos (120°—a)$$
$$4. \quad e^3 = e^2 — e^1$$

When a is unknown, it can be calculated from the relations of any two of the potential differences. For from 1 and 2 it is calculated that :—

$$\tan a = \frac{2\ e^2 — e^1}{e^1\ \sqrt{3}}$$

and using (4)

$$\tan a = \frac{e^2 + e^3}{(e^2 — e^3)\ \sqrt{3}}$$

The method has been utilised by Einthoven and others, to measure the deflection of the electrical axis during the acts of breathing (see Chapter IX). To take a simple example, if the relative values of the potential differences in leads *II* and *III* at a given time instant* are 10 and 5, respectively ; then, from the formula

$$\tan = \frac{e^2 + e^3}{(e^2 — e^3)\ \sqrt{3}}$$ we obtain

$$\tan = \frac{10 + 5}{10 — 5\ \sqrt{3}} = \frac{3}{\sqrt{3}} = 1{\cdot}732$$

Now 1·732 is the tangent of 60°. The electrical axis of the heart at the required moment is thus calculated to be 60° to the horizontal. That is to say on this occasion it lies parallel to the line of lead *II*.

Simple geometric examples.—Einthoven's method, which is employed for exact calculation, may be illustrated and rendered clearer by simple geometric examples. Unless it is fully appreciated, it is not possible to grasp the meaning and significance of those differences in the heights and directions of the deflections as they customarily appear in the three body leads.

As a first illustration, let us suppose that the electrical axis corresponding to a particular phase of the heart's cycle (say that at which *R* is inscribed in the electrocardiogram) makes an angle of 60° with the line *RL* (Fig. 58*a*).

* Accurate standardisation of the curves is imperative in this work and the only certain way of maintaining accuracy is to chart all three leads and to ascertain, as a preliminary, that they obey Einthoven's rule ($e^1 + e^3 = e^2$) at all time phases.

We suppose that at this phase of systole the electrical axis is in the line of the lead from the right arm to the left leg. The electrical potential (E) whose axis has this direction is represented upon the side of the triangle according to its inclination to these sides. It is fully represented by e^2 along $R\ F$, for the axis is parallel to $R\ F$; it is represented to a lesser extent (by e^1 and e^3) along $R\ L$ and $L\ F$, because it is inclined to each of these sides of the triangle; but as it is inclined equally, namely at an angle of 60°, to these two sides $R\ L$ and $L\ F$, the representation along these two sides is equal. E and e^2 are equal. E has double the value of e^1 and double the value of e^3, e^1 and e^3 are equal, and $e^2 = e^1 + e^3$. Thus, in this example, if R possessed a value of 10 in lead II, it will possess a value of 5 in both lead II and lead III.

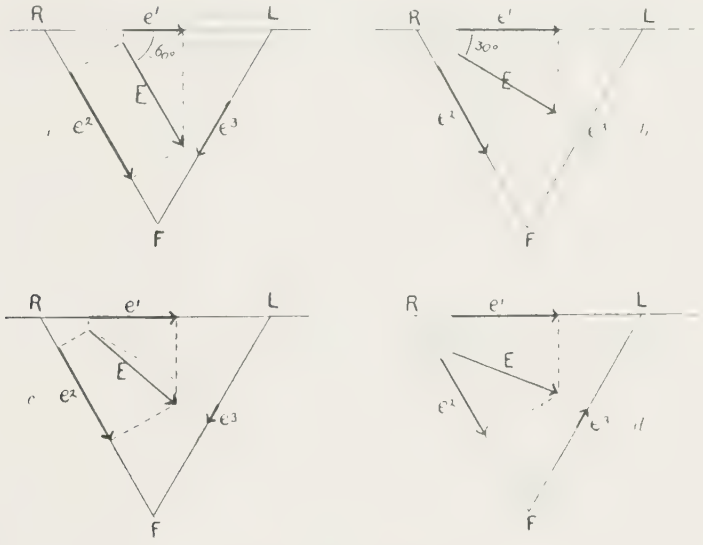


Fig. 58*a*, *b*, *c* and *d*.

In the second example, (Fig. 58*b*), the axis is represented as inclined to $R\ L$ at 30° and represents, let us suppose, the electrical axis, while T is inscribed in the electrocardiograms. In these circumstances the difference of potential will be equally displayed along $R\ L$ and $R\ F$ (by e^1 and e^2); but as the axis is at right angles to $L\ F$, it will fail to be represented along $L\ F$. Thus, in this example, if T has a value of 5 in lead I, it will have a value of 5 in lead II and will not appear in lead III. But suppose that, as in Fig. 58*c*, the electrical axis of Fig. 58*b* has moved slightly in a clockwise direction; in these circumstances the potential difference along $R\ F$ (namely, e^2) will increase somewhat, for the axis and $R\ F$ are brought closer into the same line; on the other hand, the potential difference along $R\ L$ (namely e^1) will decrease, for the angle between the axis and $R\ L$ is brought nearer to a right

angle. Moreover there will now be some slight difference of potential along *L F* (namely e^3). Thus in this third example, *T* will be greatest in lead *II*, slightly less in lead *I*, and inconspicuous in lead *III*.

Finally, let us suppose that instead of moving slightly in a clockwise direction, the axis of Fig. 58*b* has moved an equal degree in the reverse direction. In this case, (Fig. 58*d*), the potential difference along *R L* will increase while that along *R F* will diminish. There will also be a slight difference of potential along *L F*, but the current flow will be reversed in direction.

Thus in the fourth example, *T* will be tallest in lead *I*, somewhat less tall in lead *II*, and in lead *III* it will appear as a small inverted deflection.

As in all these instances when the values of e^1 and e^3 are added or subtracted according to their signs they will be found to equal e^2, so corresponding deflections in the electrocardiograms of the three leads will be found to have this simple relation, providing that these curves are accurate.

To calculate approximately the angle at which the electrical axis stands to the horizontal (*R L*) at a given phase of the cardiac cycle the relative values of the deflections at this phase must be known. These values may be accepted if they bear to each other the relation $e^2 = e^1 + e^3$. The direction of the axis may then be drawn so that, when it is projected to the three sides of an equilateral triangle its projections upon the three sides have approximately these relative values and the correct directions.*

Rotation of the electrical axis.

To apply the method described to the analysis of the dextrocardiogram and levocardiogram (*475*), we may first use an experiment upon a dog as an illustration. Curves are taken from the usual three leads subsequent to section of the right division of the bundle, and each of these curves is written simultaneously with a constant standard curve to ascertain their precise time relations to each other. The three curves are charted above one another. They are known to be standardised accurately if they obey Einthoven's rule; the values in lead *I* and *III* being together equal to the value in lead *II*. The electrical axes are now calculated for each abscissa of 0·01 or 0·005 sec. and tabulated, (see numbered lines of the network in Fig. 59 and the angle diagram beneath).

Now a given electrical axis is taken to correspond to the average direction in which the excitation wave is tending to move at the corresponding instant

* For a further discussion and illustration of the electrical axis reference may be made to page 127.

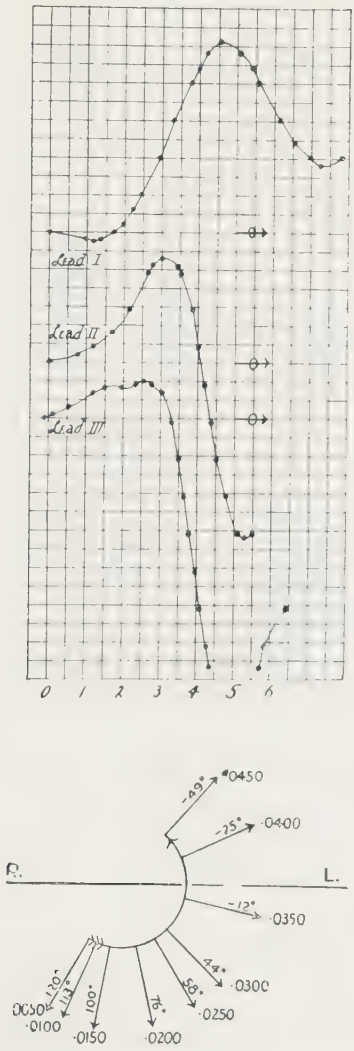


Fig. 59. (*Phil. Trans., 1916, B., CCVII., 260, Part III, Fig. 4.*) A chart, showing the initial phases of the levocardiogram in a dog in which the right division of the bundle had been destroyed. The figures below the chart mark successive hundredths of a second lines. (Ordinates, 1 cm. = 1 millivolt; abscissæ, 1 cm. = 0·02 sec..)

Below is a diagram showing the direction of the electrical axis in each succeeding 0·005 second. At ·0050 second after the beginning of the curve the axis makes an angle of 120° with the horizontal; during the next ·0400 sec. (*i.e.*, up to ·0450 sec.) the axis rotates in a regular anti-clockwise direction until it reaches —49°.

of time.* This average direction changes from instant to instant during the progress of the cardiac cycle. In the case of the dog's levocardiogram, here represented, the angle, formed by the electrical axis with the horizontal line, moves gradually from 120° to —49° in a regular anti-clockwise fashion. It indicates that the average direction in which the excitation wave tends to move in the left heart changes similarly while the initial phases of the levocardiogram are inscribed. This change is brought about by the terminal arrangement of the left division of the bundle. As this division lies on the left side of the septum it transmits the wave into the septum from left to right in the animal; as the arborisation passes to the apex, it transmits the wave from above downwards; as it reaches the lateral wall, it transmits it from right to left with an increasing upward inclination. These are the successive routes which, on anatomical grounds, the excitation would be judged to follow in its passage to the surface of the ventricle. The electrical axis indicates the general direction of these routes at corresponding instants of time.† The distribution of the excitatory process in the dog's left ventricle is approximately as it is depicted in Fig. 60, and the deflections of the levocardiogram are a natural effect of this distribution. This hypothesis is in harmony with direct readings from the surface of the left ventricle, comparable examples of which are inserted upon the present illustration.

The distribution to the dog's right ventricle is less easy to unravel, on account of the free bridging of the cavity by conducting tissue, and the meaning of the dog's dextrocardiogram is, as a consequence, less clear. The distribution, as ascertained electrically, is approximately as it is shown in Fig. 60. Without entering further into the details of the spread of the excitation wave in the dog's ventricle, we may proceed to apply the knowledge which we have gained from it to our study of the human heart.

Certain curves are obtained in clinical practice which for good reasons (see succeeding chapter) are held to represent lesions of the main divisions of the *A-V* bundle. I have chosen from my collection sets of curves from

* This statement, though perhaps not strictly accurate, is a convenient expression. The electrical axis represents of course the distribution in space of certain potentials at the instant of time to which the axis corresponds. These potentials and their distribution are governed by the events occurring in the muscle. A given region (*P*) of the ventricular muscle becomes active and therefore relatively negative to a second region (*D*) through which the excitation wave will shortly travel. The electrical axis is an imaginary line joining *P* to *D*, it is actually the line of the greatest difference of potential *at a given instant*; but since the excitation wave will travel in the natural course of events from the point first relatively negative to the point relatively positive, the line of the electrical axis is in practice the same as the line along which the excitation is tending to move. If one or more excitation waves are moving in several directions simultaneously, the electrical axis is governed by a number of potentials and resolves itself to an imaginary line which represents the sum of these potentials according to their distribution in space and their individual magnitudes. The axis continues to correspond to the direction in which the excitation wave is tending to move, though it now indicates average movement.

† Actually the electrical axis is, of course, governed by the distribution of the regions of relative negativity and positivity (see last footnote).

two patients, and have charted the curves from leads *I*, *II* and *III* in both instances. These curves are held to represent deficient conduction in the right division (Fig. 61*a*) and of the left division of the bundle (Fig. 61*b*) respectively.*

The electrical axes have been calculated for the two sets of curves and the rotations of the axes are represented in the annexed diagrams. The result is most instructive, for it shows that in the case of the human ventricle the rotation is simple and uniform in both ventricles; a uniform anti-clockwise

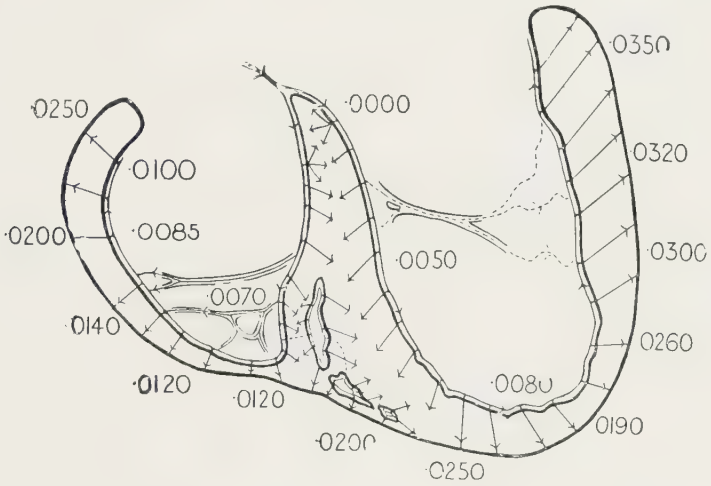


Fig. 60. (*Phil. Trans., 1916, B., CCVII., 262, Part III, Fig. 5.*) A natural size diagram of the dog's ventricle seen in section, and illustrating the direction taken by the excitation wave in the two chambers. These directions are indicated by the arrows and the numbers are inserted opposite endocardial and pericardial points to indicate the approximate times in seconds at which, after the commencement of the electrical disturbance, the excitation wave arrives in different regions of the muscle. The basis of the calculation in inserting these numbers has been the actual readings by the method described in Chapter VII. These readings and the times at which the electrical axis assumes corresponding directions (see Fig. 59) are in close accord; approximately 0·01 second should be added to each number in the present illustration (the time assumed for the full development of the electrical discharge) to render this harmony almost perfect.

movement in the left chamber (Fig. 61*a*) a uniform clockwise movement in the right chamber (Fig. 61*b*). In man there is no bridging of the cavities by free strands of Purkinje substance,† and the axes of the electrical disturbance moves in a regular fashion; on both sides the excitation wave spreads first into the septum, moving down and towards the right on the left

* To place such human curves in proper time relation to each other they are moved until they fit; *i.e.*, until on any vertical line, the value in lead *II* is equal to the combined values in lead *I* and lead *III*. This procedure is necessary, and has been adopted in the case of these charts where no standard curve has been taken to facilitate orientation.

† Possibly there are exceptions to this statement (see Griffith, *Proc. Anatom. Soc., July, 1899, Journ. of Anat., Vol. XXXIV*).

side, and down and towards the left on the right side ; later it moves directly downwards at the apices of the ventricles ; later still, it moves horizontally outwards on both sides through the lateral walls of the heart ; finally it moves upward and outward as it reaches the base of the heart. By comparing

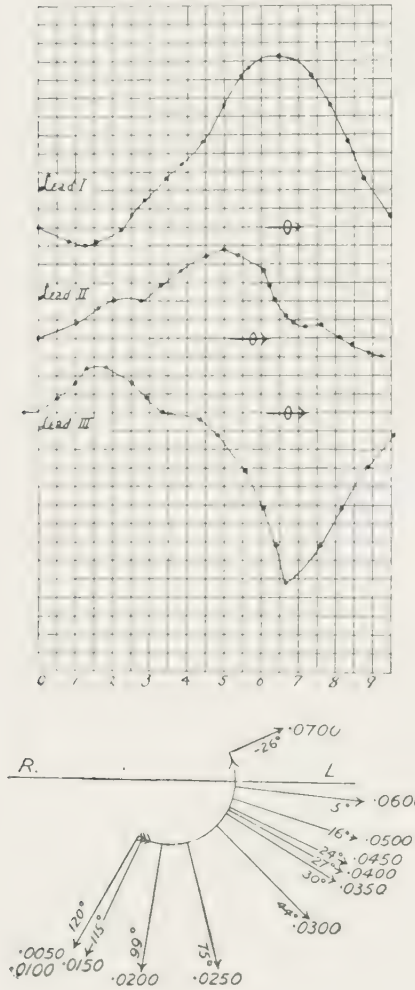


Fig. 61*a*.

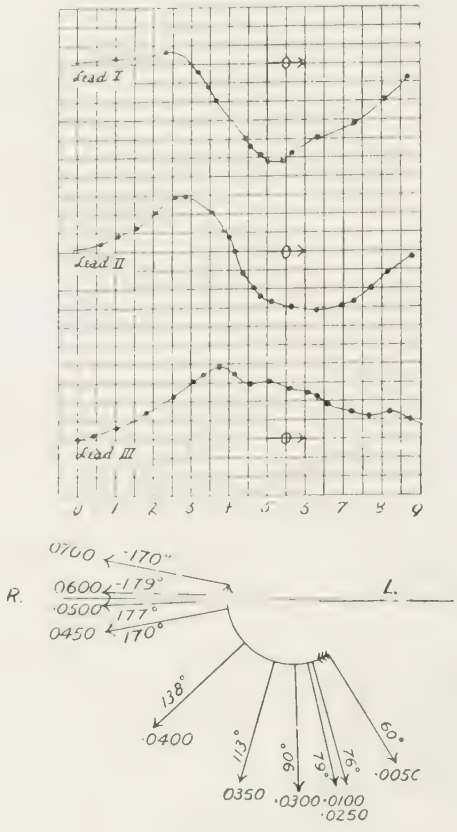


Fig. 61*b*.

Fig. 61*a*. (*Phil. Trans., 1916, B., CCVII, Part IV, 284, Fig. 1.*) A chart of the initial phases of human electrocardiograms, taken from a heart presenting signs of deficient conduction in the right division of the bundle. Ordinates, 1 cm. = 1 millivolt ; abscissæ, 1 cm. = 0·02 sec. A diagram showing the angles of the corresponding electrical axes and the rotation.

Fig. 61*b*. (*Phil. Trans., 1916, B., CCVII, Part IV, 284, Fig. 2.*) A similar chart and diagram, taken from a heart presenting signs of deficient conduction in the left division of the bundle. Ordinates and abscissæ as in Fig. 61*a*.

a number of human electrocardiograms, charted and analysed in this fashion, and by utilising as a basis the fuller data obtained in experiment upon the dog, it becomes possible diagrammatically to represent the distribution of the excitatory process in the human ventricle, recording against the endocardial and pericardial surfaces of the heart, as represented in section, the approximate times at which the electrical disturbance reaches the various regions of the musculature during the normal ventricular cycle. And this may be done, as in Fig. 62, with some pretence to accuracy.

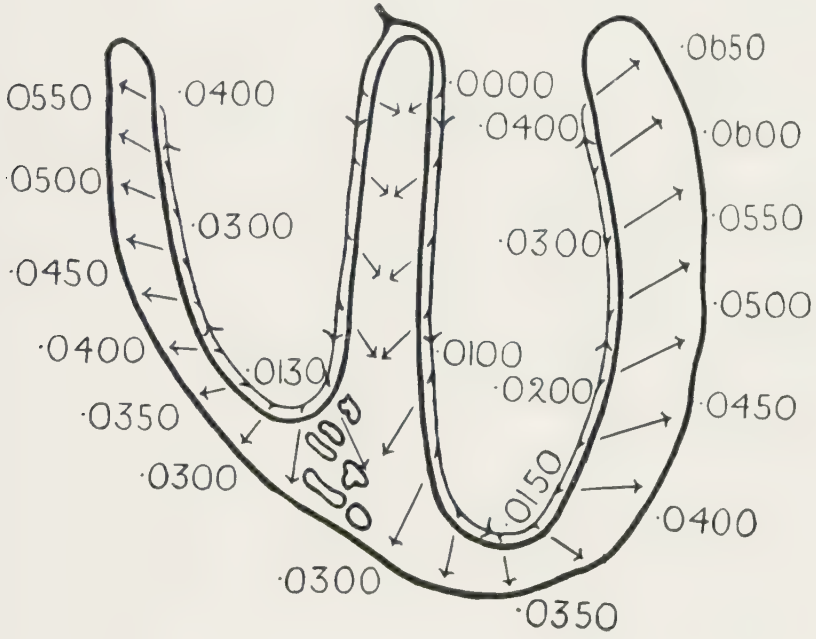


Fig. 62. ($\times \frac{1}{2}$.) A diagram of the human heart as seen in section. It represents the author's considered view of the directions in which the excitatory process spreads in the human ventricle, and the times in seconds at which, after its commencement in the ventricle, this process first reaches the various regions of the ventricle.

Now if the general conclusions here set forth be accepted, and the considerable body of evidence in our possession at the present time is in their favour, they satisfactorily explain a number of phenomena. Some of these may be briefly sketched at once and elaborated in the next chapter. According to my view, the human electrocardiogram, taken from whichever lead, is a composite of left and right effects. Its earliest events are those associated with activation of the septum on right and left side ; the activity

of this part is responsible for Q* and the commencement of R in all leads. Q and the chief part of the limb R are right-sided events in leads II and III, left-sided events in lead I. S is a right-sided event in lead I; a left-sided event in leads II and III. Corresponding deflections are to be found in the dextrocardiogram or levocardiogram as the case may be. Briefly, we have an explanation of all the initial deflections of the electrocardiogram, which not only harmonises with physiological observation, but which is in complete accord with contemporary anatomical discoveries. We are also provided, as the next chapter will show, with a clear conception of those abnormal clinical curves which are associated with preponderating hypertrophy in one or other ventricle, an explanation which we have lacked hitherto.

The end-deflection "T."

Upon a written discussion of T in the electrocardiogram I enter with some reluctance;† while desiring to refrain from airing such conceptions as have passed through my mind, yet it is hardly possible for me to do so without leaving an obvious break in the continuity of this book. In the circumstances I can but refuse to commit myself further than to indicate in a general way my reflections on this subject and the basis from which, as it seems to me, further observational work might begin. Nevertheless, I fancy I have this advantage over writers who have previously taken this course, namely, that the detailed path of the excitation wave in its spread through the ventricle seems clear to me, while from them it was, at all events in large part, hidden. But there are fundamental points in respect of which we still lack definite or final knowledge, and until such knowledge is won a full explanation of the deflection T and its variations does not seem possible.

In a previous chapter the usual conception of the electrical events in a simple strip of muscle, forced to contract from one end, is represented diagrammatically (Fig. 29, page 54). Muscle, when it becomes active is known to show relative negativity to inactive muscle. In the muscle strip of the illustration the wave of activity passes from P to D, and, as I have described, when it arrives and reaches its full force at D, P and D, assume the same electrical state and remain for a variable period isopotential. But it is supposed that the point P maintains the electrical state relative to the inactive point which it assumes at its first activation, and that the point D after its own activation behaves likewise; for this

* In the amphibian and reptilian heart, in which there is no septum, Q is not seen in the electrocardiogram. It has been asserted repeatedly that the frog's electrocardiogram presents identical initial deflections to these of the human electrocardiogram. This is not the case, for in the former Q is always absent.

† In speaking in this chapter of T, I mean to refer only to the end-deflection in the electrocardiogram and not to end-deflections in direct leads from the heart.

supposition is compatible with a continued isopotentiality of the two points* and explains the final deflection of the string. It is supposed that the electrical disturbance is maintained at its height at both points and that isopotentiality is continued, until the excited state of *P* begins to decline ; a deflection of opposite sign to the original deflection is then produced. Although this hypothesis is very possibly in the main correct, yet there is no proof that the electrical state of an active muscle point is constant in degree throughout any prolonged period of the systole. It occurs to me that the *full* change, which is undoubtedly manifested by muscle as it enters the active state, may be a more transient effect than is commonly supposed and that Burdon Sanderson's classical diagram (*685*), which is reproduced in modified form in Fig. 63, may fail, in that respect at least, to convey a true impression of what happens. It seems quite possible that the electrical change in the initial phases of activity may be of a different order quantitively to that of the late stages and that the plateau of the diagram should not be represented as the highest point reached. In testing active and uninjured muscle we obtain no measure of the electrical change at one point ; it is the difference of potential between two points, the one active, the other as yet inactive, which we observe. Moreover, we have no measure even of the approximate duration of electrical activity at one point. We assume that the duration is from the instant when the excitation wave arrives to near the termination of the end-deflection : but even this assumed interval can be ascertained only approximately, for the end-deflection subsides and reaches the base line very gradually.

In expressing the following view of *T*'s constitution, I do so subject to these reservations which I have touched upon.

The state of activity in a simple muscle strip may be described as comprising three phases :—(1) a *stage of invasion*, a stage during which the excitation wave is spreading ; (2) a *stage of possession*, or a stage in which isopotentiality is found as a rule between all parts of the muscle and during which activity is *supposed* to be maintained in full force ; and (3) a *stage of retreat*, during which activity is thought to subside in the order in which it came. With the first stage as it applies to the heart I have dealt fully and in so far as this portion of the hypothesis is concerned we may rest content that it is sound ; we now know from direct observation, what has hitherto frequently been supposed, namely that the initial phases of the electrocardiographic curve correspond to the spread

* This period of isopotentiality is not always to be observed in direct leads even in the frog and tortoise heart, where its production would seem to be especially favoured by the long duration of the contraction. Actually there may be during the middle period of systole an almost constant deflection of the string to one side of the zero line. Further, before the isopotential state is reached, there is often in direct leads from the heart, a second deflection in an opposite direction to the first.

The isopotential period in human electrocardiograms is usually brief, and in many cases non-existent.

of the excitation wave and that the presence of excited areas, alongside of unexcited areas, disturbs the balance of potential. It is in regard to the second and third stages that there is uncertainty; there are some writers indeed who believe that when the last phases of the electrocardiogram (*T* deflection) are inscribed the lack of potential balance is due, not to the decline of the same excitation process which in its spread produced the

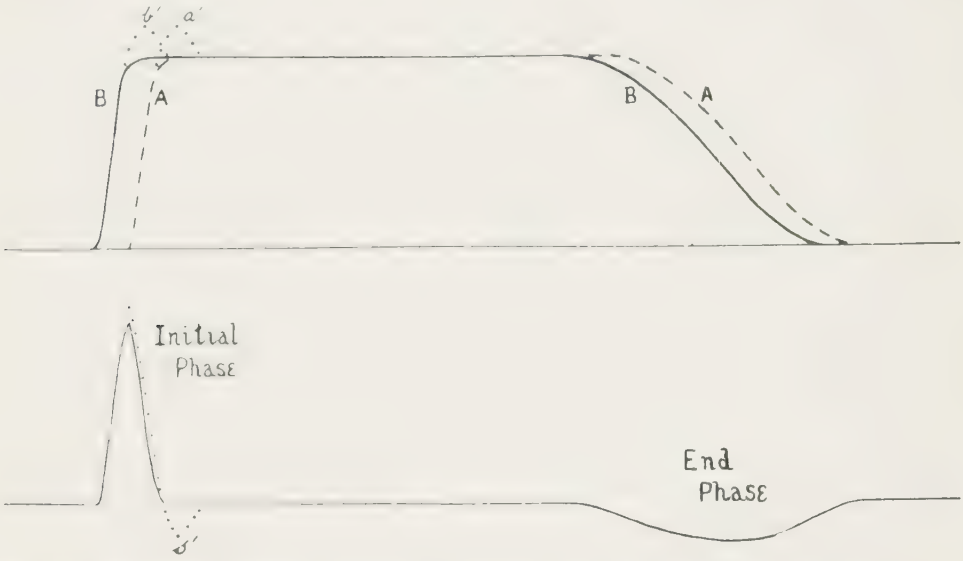


Fig. 63. Sanderson's diagram (modified) to explain the two chief deflections given by the cold-blooded ventricle. The actual curve obtained from the heart, and shown below in the diagram, depends upon differences of potential at the two contacts (B = basal; A = apical); these are expressed by corresponding differences between the two curves (B and A) measured vertically. The two curves B and A are drawn of identical duration and form, but B is placed somewhat earlier in the figure, to represent the earlier arrival of the excitatory process at the heart's base.

In Sanderson's diagram the curves B and A rise abruptly until they reach the level of the plateau and do not rise above this level; the rise represents the stage of *invasion*. The plateau is represented by a long continued horizontal line; this represents the stage of *possession*. Finally, the curves B and A descend gradually to meet the zero line; this represents the stage of *retreat*.

The curves are theoretical; we have no direct knowledge of their forms. It is possible that the highest point of each curve is reached, not in the plateau, but in the stage of invasion, as indicated in the dotted lines of the diagram. The presence of these early summits would alter the resultant curve as indicated by the dotted line in the lowest curve.

initial deflections of the curve, but to the decline of a totally distinct process, namely, the *contraction* of the muscle (*160*, *325*, *682*, *695*).

The chief source from which the last view seeks support is that the end deflection is said to be more pronounced the stronger the ventricular contraction, and that where there is no visible contraction *T* may disappear while the initial phases persist (*325*, *682*). But the waxing of *T* in the

electrocardiogram with the strengthening of contraction is not without notable exceptions; in alternation of the heart (see Chapter XXXIII) T frequently alternates in height, and the large T may correspond with the weaker ventricular beat. The view that T is unrelated to the excitation process is not supported by observation; on the contrary there is some direct evidence that they are intimately connected. Thus, Mines (*555*) observed what he was convinced was complete cessation of contraction in the heart, while the galvanometer showed strong regular deflections of the usual general form. This result he obtained by perfusion with a calcium-free Ringer solution, and he published a figure showing a conspicuous end-deflection. Other evidence we shall examine directly.

Misconception has not infrequently arisen in that many writers appear to have tacitly or openly regarded the end-deflection as a manifestation peculiar to the ventricle or to a particular part of it. This is not the case; a similar end-deflection is found in records of the auricular systole, though, as the deflection is a slight one and usually buried in the initial phases of the ventricular curve, it is not often seen (*159*, *292*, *555*). A similar end-deflection is produced by the truncus arteriosus of the tortoise heart (*475*). But we may go much further than this and assert that the end-deflection is not peculiar to a single chamber of the heart or even to heart muscle; a corresponding deflection appears in the final phases of the contraction process in a strip of muscle, a fact which has been recognised for very many years; and it is a matter of indifference whether the muscle is somatic or cardiac.* The deflection comes from a lack of potential balance and this, according to the most generally accepted present day view, is attributable simply to the retreat of the excitation process. But it is to be emphasised that of this retreat, and especially of the path it pursues and of its rate of progress, we have little definite knowledge. It is considered probable that the rate of descent of the excitatory state is more gradual than is the rate of its ascent. The steepness of the intrinsic deflection is the measure of the latter in curves taken direct from the muscle. The rate of descent is judged from the rate at which the end-deflection declines. The initial deflections arise abruptly, T fuses almost imperceptibly with the base line; so it is felt that the excitatory process dies away slowly, and this view is expressed in such diagrams as that of Fig. 63.

A simple strip of muscle gives two deflections, an abrupt deflection in one direction and a more leisurely one in the other. These two deflections correspond to the stage of invasion and of retreat, and for that reason, although they both correspond to a movement in one direction through the muscle, it is the rule that *they are opposite in sign* (see Fig. 29, page 54).

The more detailed application of this view to the mammalian electrocardiogram may be introduced by citing the relatively simple ventricle

* In suitable circumstances it appears in both direct and indirect leads

of the amphibian or reptile. In this single ventricle the excitation wave spreads from the *A-V* ring along the almost straight muscle bands of the interior to reach the main wall of the ventricle at points midway between base and apex (Fig. 64). Subsequently it courses downwards to the apex and upwards to the base. In the simplest instance, the base is first reached and from first to last the general movement of the wave is from above

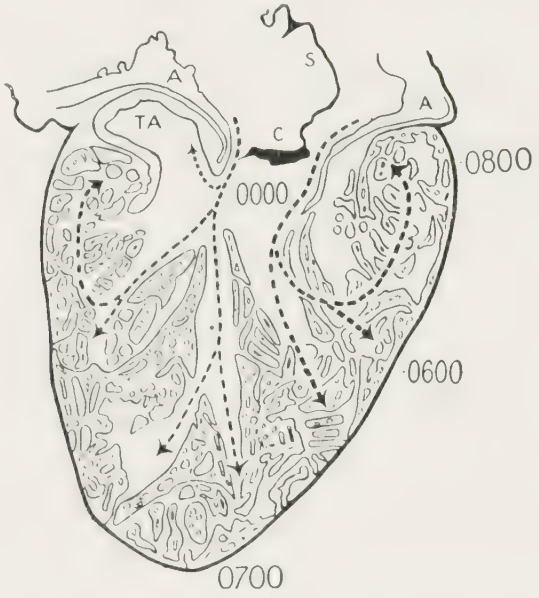


Fig. 64. (*Phil. Trans. Roy. Soc., 1916, B., CCVII., 236, Part I., Fig. 6.*) (× 5.) A diagram of the toad's heart, seen in coronal section, and of the manner in which the excitation wave is supposed to spread through it. *A* = auricular muscle passing into *A-V* ring. *C* = endocardial cushion. *S* = auricular septum. *T.A.* = commencement of *truncus arteriosus.*

downwards. The electrocardiogram opens, as we should expect, with a single prominent upward initial deflection *R* in leads *II* and *III* (representing an electrical axis of approximately 90°). As in this example the last point to become active is the ventricular apex, so it is to be expected that the apex will form the last point in the retreat. In other words it is assumed, though it is not proved, that the order of retreat is the same as the order of invasion. As in the case of the simple strip of muscle, it is to be anticipated that the end deflection (*T*) will have the reverse (*i.e.*, downward) direction to the initial deflection (*R*). This is found actually to be the case.* But *T* is not always a downward deflection in the amphibian electrocardio-

* It is to be pointed out that this and subsequent statements applies to the *electrocardiogram*, a curve obtained from the heart by contacts placed on the surrounding tissues. I am not speaking of the *electrogram*, which, taken by direct leads, chiefly comprises the intrinsic deflections from small areas of muscle in immediate contact with the small electrodes.

gram ; more often it is upright ; indeed, this seems invariable when the heart is little disturbed. In such curves, in my experience, *R* does not stand alone, but is followed by a distinct downward deflection *S* (representing an electrical axis of approximately —90°), and the ascertained order in which the excitation wave spreads shows a corresponding difference. In such examples, although during the greater part of the invasion the average direction of spread is downwards* through the heart, the last points to be activated are discovered by direct observation to be at the extreme base and the average direction of spread at the last is upwards (as in Fig. 64). If we assume the same order of retreat, the base will leave its last impression on the electrocardiogram and the upright *T* is attributed to this dying activity.†

According to this view both *S* and *T* in the amphibian and reptilian electrocardiogram are basal effects, the one produced in the stage of invasion, the other in the stage of retreat. They are therefore opposite in direction, *S* being downward and *T* upward. The rule may be formulated for these electrocardiograms *that the direction of the end deflection* (*T*) *is opposite to that of the last initial deflection.* That is precisely what we are led to expect from the simple muscle curve, and from the hypothesis which attempts to explain it. The rule may not be absolute, but it is nearly if not quite so. Personally I have seen no exception to it. Thus, in the cold-blooded heart, beating naturally, we possess evidence that the direction of *T* is related to the original path of spread of the excitation process. That is of much importance.

Is there a similar relation in the mammalian electrocardiogram ? In the human electrocardiogram, of which we have most knowledge, it is the rule to find an upright *T* if the last deflection of the initial group is downward. It is also the rule that when *T* is a downward deflection, the last of the initial deflections is upright. There appears to be a similar relation to that obtaining in the cold-blooded heart, though it is not so rigid, for an upright *T* is not uncommonly seen in curves which present no *S* in the initial phases. Thus, there is in the human electrocardiogram sufficient evidence of a close, though not constant, relation between the direction of *T* and the form of the

* When the average direction is downward, the points of relative negativity (or activity) stand more closely related to the base of the heart, while the points of relative positivity (the inactive parts of the heart through which the wave will subsequently spread) stand more closely related to the apex. I again use direction of spread as a convenient expression, knowing that technically it may be open to objection (see footnote, page 104).

† Thus we come in modified form to the view expressed by Gotch (*230, 231*), that, in the naturally nourished and undisturbed heart, activity is first developed in a region related to the base (though not at the actual base as he supposed), that the first spread is to the apex, and that subsequently there is movement towards the base. The movement is not, as Gotch supposed, a relic of the *S* shape of the embryonic heart, for the excitation wave does not spread as a continuous wave from base to apex with a return to base ; originally flowing downward in the inner musculature, the wave proceeds downward and at the same time is reflected up towards the base. If it reaches the actual base a little later than the actual apex, and this is the rule, the final unbalanced movement is upward at the base.

initial group of deflections Q, R, S; the relation is particularly to the direction of the last initial deflection, as in the case of the amphibian and reptilian heart.*

In the illustration of the simple muscle strip, the deflection produced in the stage of retreat is opposite in direction to that of the stage of invasion. We might therefore be led to expect that the final and initial phases of the human electrocardiogram would show equal complexity, but that the direction of the several end-deflections would be the reverse of the initial deflections. But there is at least one good reason why the end-deflections should be of simpler form than the initial deflections; if we are correct in supposing that the decline of the excitation process is more gradual than its ascent, we should expect the final deflections to blend with one another: we should also be led to anticipate that the current produced in the last

* The directional relation of the end-phase and the initial phase would be emphasised, if a corresponding relation were found in beats forced by stimulation from the ventricle. Now, in so far as the electrocardiogram of the mammalian heart is concerned, this corresponding relation is discovered. A forced beat, whose initial phases end with an upright deflection, has a downwardly directed end-phase; a forced beat whose initial phases end with a downward deflection has an upright end-phase; thus, it is shown clearly that the end-phase in these curves varies according to the direction of the original spread (see Fig. 170, page 215). But an experiment by Samojloff (*682*) seems for the moment to be interpretable in the opposite way. Leading from the *base and apex* of the cold-blooded heart and recording natural beats or beats forced from various parts of the ventricular surface, he finds that while the initial phase varies in direction, the end phase is constant in direction. Mines (*555*) interprets this observation as meaning that the excitation process constantly lasts longer in a particular region of the heart, independently of the original order of excitation. I am not convinced that this explanation is valid. Samojloff's experiment should be repeated with the use of indirect leads; these would in all probability yield a different result, as they do in the mammalian heart. As Seemann (*695*) has shown, the Samojloff phenomenon is not constant even in direct leads. The interpretation of curves taken by means of direct leads from the heart constantly gives rise to difficulties; these curves contain much that is mysterious. I am prepared to grant that Samojloff's experiment requires explanation, and that I am unable to give that explanation. His observation and the one which I shall cite will presently form a chief reason why a full acceptance of any present day hypothesis is impossible.

In direct leads from the heart, in which the two contacts are very close together, an end phase always appears (*51*). It appears independently of the region of the lead, thus clearly showing that there is a disturbed balance even in the shortest strip of ventricular muscle towards the end of systole. The finding of this end-phase universally over the ventricle, however, does not forbid the interpretation of T in an axial and indirect lead as mainly a basal or apical effect according to its direction; for the end-phase of the direct lead is not the same end-phase as T of the electrocardiogram, though the cause of the first may be comprised in the cause of the last. This *appearance* of T in all direct heart leads is not more mysterious than the appearance of a similar deflection as a final effect in all strips of muscle which pass through the contraction process. Neither is the *variable direction* of the end-phase, in different direct leads in which two contacts are placed on the ventricle, an essential difficulty. The chief difficulty is to explain why the end-phase varies in its direction in direct leads, when one contact is placed on different parts of the ventricle while the other is maintained on a constant point on the body wall. This is the case in the cold and warm-blooded heart and I have been unable to determine what influences the direction of the end-phase in these curves. The upright end-phase or downward end-phase, as the case may be, is not particularly associated with one region of the ventricle, neither does it show constancy of direction for one ventricular area from animal to animal. This phenomenon like that witnessed by Samojloff lacks explanation at the present time.

phase of the retreat would leave a relatively strong impression upon the curve,* for at this stage it would be less opposed by conflicting currents. Thus, on theoretical grounds, we might expect a relatively simple form of end-curve, but an end-curve whose direction was especially influenced by the last phase of the retreat and therefore especially related to the last phase of the stage of invasion.

It should be understood that a quite constant and simple relation between the initial phases and the terminal phases of the curve will appear only if the path of retreat and the rate of retreat is exactly or almost exactly the same as the path of invasion and the rate of invasion. To put the same matter in a different form, this constant and simple relation will appear only if the duration of the excitation process† in muscle substance is quite uniform in all parts of it. Whether there is ever quite such uniformity we do not know and at present there seems no means of determining, though *a priori* it seems improbable. We might assume that as the muscle structure is similar in different regions of the ventricle, the duration of the excitation process is the same in different regions; but such an assumption would be at the best precarious. It is well within the bounds of probability that the duration varies in different regions under several influences, for example, nutritional or nervous influences. A simple experiment probably illustrates this very point. If heat is applied to the apex of a heart which in the base-apex curve or electrocardiogram yields a downwardly directed end-deflection, then this deflection becomes upright without any material change in the initial phases‡ (Bayliss and Starling, Mines, etc.) (*27*, *136*, *555*). This reversal of the end-deflection (T) may be explained if we suppose that heat, by quickening the processes at the apex, speeds the retreat in this part of the heart, rendering the basal parts relatively electronegative to those of the apex at the last.

The initial phases of the electrocardiogram are much less liable to change of form and direction than is T in a given instance. This rule conforms to the experience of all workers, and there are many conspicuous examples of it. One noteworthy example, namely, the effects of local change of temperature, has been discussed. Another almost equally prominent example is found in the effect of nerve stimulation. Stimulation of the vagus or sympathetic nerves has profound effects on the end-deflection T; but the effects on the initial deflections are relatively slight (*664*, *682*). Many poisons act in a similar fashion. As Cohn and his fellow-workers (*66*, *67*) have shown, an early effect of digitalis intoxication in the human subject is an inversion of T;

* At all events upon the last part of T, which is the most constant in direction.

† The time from which the invasion begins at a given point to the time at which the retreat leaves it free.

‡ Heat applied at the base or cold applied at the apex, produces the opposite effect on T. The experiments of Eppinger and Rothberger (*136*), in which various parts of the ventricle were frozen, are to be classed with these cooling experiments and are not to be viewed as experiments in which large parts of the wall are thrown out of action.

this effect of digitalis appears to result from a direct action of the poison upon the muscle.

To sum up these reflections, it may be said that the direction of *T* seems to be related to the manner in which the excitation process spreads in the ventricle, and there is suggestive evidence that the invasion and retreat follow more or less the same path when the heart beats naturally. If that is so, then the hypothesis, which supposes the excitation process (with its accompanying state of relative negativity) to continue throughout the whole of systole, sufficiently explains the chief experimental observations. But if this hypothesis is adopted, it would also be necessary to suppose that from time to time certain influences, such as changed nervous control or the action of poisons, may prolong or shorten the excitation process in one part of the ventricle to a greater extent than in another, and thereby promote dissimilarity between the path or rate of advance and retreat. In such fashion the curious relations of the deflection *T* under altered innervation or poisoning of the heart, might be explained.

The elucidation of *T* might conceivably be approached from a slightly different standpoint. As we have seen, the initial phases of the electrocardiogram are comprised by the superimposed effects of right and left ventricle. If we are right in assuming that the duration of the excitatory process is uniform in different parts of the heart, then *T* is also a dual effect and is to be regarded as the product of the end-deflections of right and left ventricle. Unhappily we have no certain means of ascertaining either the direction or value of the end-deflections of the true dextrocardiogram and levocardiogram. There is some reason to believe that the end-deflection of the human dextrocardiogram and levocardiogram* are opposite in direction to the last and chief initial phases in these curves; thus, in lead *III* and usually in lead *II* the end-phase of the dextrocardiogram would be directed downward, and in the levocardiogram it would be directed upward; whereas in lead *I* the directions would be the reverse. In that case the upright *T* of the normal electrocardiogram would be attributable to a preponderance of the left ventricular effect in lead *I* and of the right ventricular effects in lead *II* and *III*. But the evidence is no more than suggestive and cannot in this place profitably be pursued further.†

Alterations in the value or direction of *T*, both of which are commonly seen in cases of heart disease, have not as yet received any adequate explanation.

NOTE.—The following additional publications may be consulted for further discussions and views on the constitution of the electrocardiogram (*117, 118, 325, 392 and 573*).

* I am speaking of the directions of the real end-deflections and not of end-deflections which appear when the left and right bundle branches are divided; for these, as I have explained, are not reliable indications.

† Experiments in which one or other ventricle is removed and the resultant curve observed (*138*), are perhaps too crude to possess material value.

Chapter IX.

Aberrant Contractions and the Electrocardiograms of Hypertrophy, etc.

In Chapter VI, the electrocardiograms portraying defects in the chief divisions of the dog's *A-V* bundle have been described, and it has been shown that the changed form of the curves results from altered distribution of the excitation wave in the ventricles. In the normal heart beat, the spread to each ventricle is through the bundle divisions and their arborisations; the spread is simultaneous in the two ventricles in these circumstances. On the other hand, when one division of the bundle is cut across, the spread is at first confined to the contralateral or unaffected ventricle and, so far as this ventricle is concerned, it is normal; later, the spread is to the affected ventricle and in this the distribution is wholly abnormal. Thus the spread of the excitation wave to the ventricles, considered together, is abnormal by reason of a defect in a chief distributing channel; consequently I term the resultant contractions *aberrant*, for they are the product of impulses which have gone astray.

In the last chapter certain human curves were used on the assumption that they express similar aberration in the human heart. The reasons for regarding these electrocardiograms as representing aberrant systoles may now be set forth.

It will be shown in a later chapter that defective conduction through the *A-V* bundle is not an uncommon incident in cardiac disease; and histological studies have proved that the *A-V* conduction system, including the main bundle and its branches, is particularly susceptible to certain forms of degenerative and inflammatory change; these injuries fall upon the conducting system selectively. The co-existence of discrete lesions of similar type in the main bundle and in one or both of its chief divisions has been observed on many occasions; in other and numerous instances a single lesion has been found to involve the main stem and one of its two offshoots. Therefore, it is natural to anticipate that curves representing aberrant contractions will be obtained from the human subject. Now in any large collection of clinical electrocardiograms, a number of peculiar curves, having the distinctive forms of those used in the last chapter, are to be found and these same electrocardiograms show, as often as not, defective *A-V* conduction. The frequent association of *A-V*

block in its various degrees with the curves which we are now considering, may be taken as a first and important evidence of their origin.

It is to be hoped that ultimately, a sufficient number of cases may be adduced in which these curves have been obtained and in which corresponding lesions have been seen microscopically; but at the present time, owing to the difficulty of collecting such material, only a few instances can be quoted. In describing the curves now spoken of as levocardiograms, Eppinger and Stoerk (*140*) regarded a sclerosis of the septum, involving the right division of the bundle as the responsible lesion; they had indeed diagnosed such a lesion during life. In a case examined by Cohn and myself (*72*), in which curves of the same kind were published, a lesion was found in the right division.* A second example has recently come within my own experience, the expected lesion being discovered by Dr. Butterfield who examined the heart for me. On the other hand, in several hearts examined for me (*75*) by Cohn,† no such lesions were found. Briefly, there are instances in which the expected lesions have been discovered, and these support our conclusion. There are also instances in which no lesion has been seen and these do not materially weigh against it‡; similar discrepancies between functional damage and anatomical discontinuity are frequent when *A-V* block has been discovered, discrepancies which will be described in the proper place.

But the strongest reasons for regarding the human curves as representing defects of the bundle divisions is still the comparison of the human curves with those obtained experimentally.

Curves held to represent defects of the right division (the human levocardiogram).—The human levocardiogram (Fig. 65*a*) comprises three deflections in lead *I*. An inconstant deflection Q' is succeeded by a tall and broad summit R' (usually notched) and this is followed by a deep and rounded depression T'. In lead *III*, the first deflection is a small summit R' and this is followed by a broad and extensive dip S', which is often notched, and by a large and rounded elevation T'. The curves of lead *II* are variable, and having smaller amplitudes are of less consequence; usually they are similar in outline to those of lead *III*, or consist of a number of small phases. The amplitudes of the chief deflections in leads *I* and *III* exceed, and often greatly exceed, those of natural electrocardiograms. The initial phases

* Associated with a lesion in the main stem and complete *A-V* block.

† Who has reported upon them in our joint names. The electrocardiograms in these patients were in each instance characteristic of defective conduction in the right stem. The clinical notes (Hearts 42, 44, 73 and 91 of Cohn's report) are reported by Carter (*46*), as cases 5, 18, 20 and 19 respectively. The weight of the separated ventricles in the patients are given in a separate paper of my own (*467*), and Cohn's numbers correspond to those in the column " case number " of my tables. My reason for referring to these weights is that they show that the curves were not the result of preponderating left hypertrophy.

‡ They weigh against the conclusion less in that the division in its whole length was not examined.

as a group present an exaggerated duration in all leads; the Q, R, S group in the normal human electrocardiogram has an average duration of 0·08 sec., the initial phases of the human levogram average 0·14 sec. in duration, and always exceed 0·1 sec. (*453*, *463*, *475*). It is also noteworthy that the chief deflection, R' or S' as the case may be, is always opposite in direction to the final wave, T'.

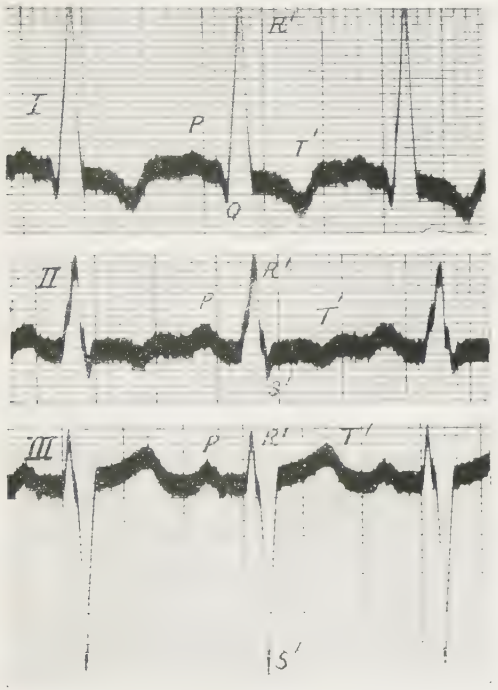


Fig. 65*a*.

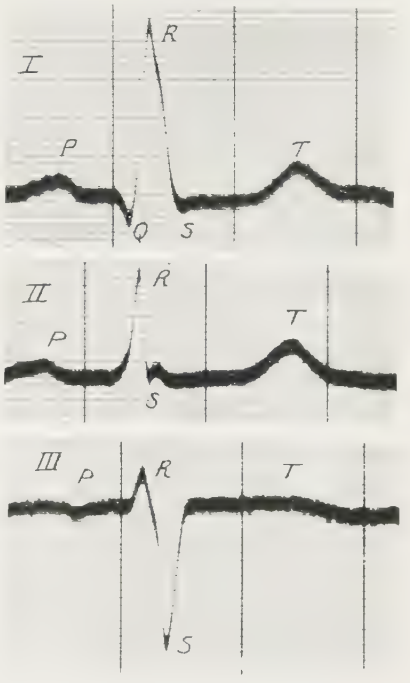


Fig. 65*b*.

Fig. 65*a*. (*Phil. Trans., 1916, B., CCVII, 284, Part IV, Fig. 11.*) Electrocardiograms from the three leads in a patient. Curves of this type are associated in the human subject with defects of the right division of the A-V bundle. Time in fifths and twenty-fifths of a second.

Fig. 65*b*. (*Phil. Trans., 1916, B., CCVII, 284, Part IV, Fig. 12.*) Human electrocardiograms from the three leads. From a case of aortic disease with great hypertrophy of the left ventricle. These curves are almost identical with those of Fig. 65*a* in their initial phases; they differ from them in their final phases (T' and T). Time in fifths of a second. These two series of curves have been specially selected for their resemblances.

Curves held to represent defects of the left division (the human dextrocardiogram).—The first curves of this kind to be described (*463*), are shown in Fig. 66*b*.* In lead I, a diminutive deflection R' is succeeded by a deep and

* The rarity of human dextrocardiograms and the comparative commonness of the human levocardiograms is to be explained chiefly by an anatomical difference. The right division has a long course as an unbranching strand, the left quickly subdivides and after the first part of its course the complete division of this portion of the conducting tract would necessitate a very extensive lesion.

broad deflection S', and an upwardly directed T'. In lead III a diminutive Q' and a tall, broad and notched R' are followed by a depression T'. The curves in leads I and III are similar to leads III and I, respectively, in levocardiograms; they are in the main diphasic, T' having an opposite direction to the chief initial deflection. The initial deflections are of long duration as compared to the normal.

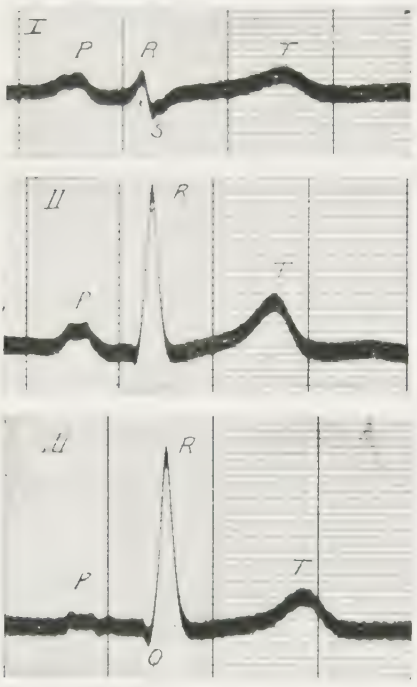


Fig. 66a.

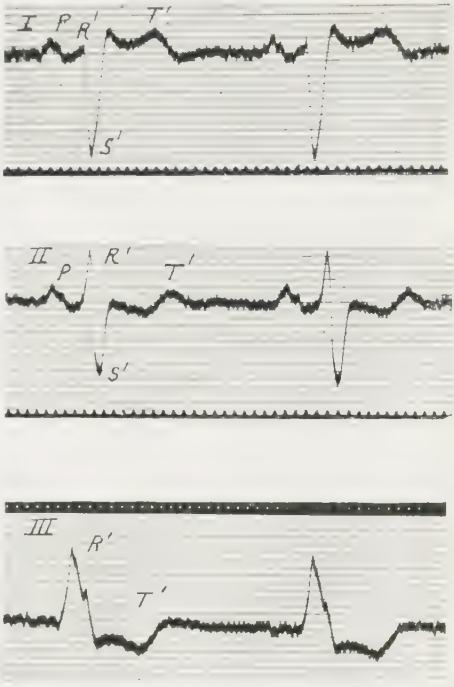


Fig. 66b.

Fig. 66a. (*Phil. Trans., 1916, B., CCVII, 284, Part IV, Fig. 13.*) Three electrocardiograms from a case of mitral stenosis; illustrating preponderating hypertrophy of the right ventricle. Time in fifths of a second.

Fig. 66b. (*Phil. Trans., 1916, B., CCVII, 284, Part IV, Fig. 14.*) Electrocardiograms from the three leads in a human subject. These anomalous curves were probably produced by a defect of the left division of the bundle. Time in thirtieths of a second.

Comparison with experimental curves.—Comparing the general features of the human curves and those obtained experimentally from the dog, we find the following features in common, whether we are dealing with levocardiogram or dextrocardiogram, and irrespective of lead:

1. The amplitude of the chief deflections is more than normal.
2. The initial phases have an unusual duration.

3. The final deflection is always opposite in direction to the chief initial deflection; each curve is, as a whole, broadly diphasic.*

If the human and canine levocardiograms are compared in detail, very close resemblances are noticed in form (see Fig. 44, page 77, and 65*a*, lead *III*). Complete correspondence between the number and directions of deflections is frequently to be found, and even the notching of the chief initial deflection is usual in the human curves.†

The dextrocardiogram of the dog and of man may present notable differences in their detail and this is to be expected, seeing that there is a difference in the distribution of the Purkinje strands to the right ventricle.

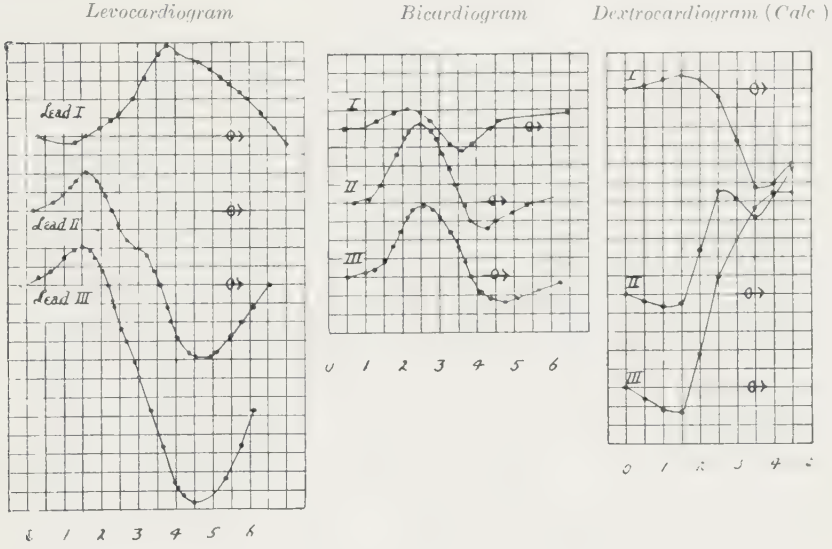


Fig. 67. (*Phil. Trans., 1916, B., CCVII, 287, Fig. 3.*) A chart showing three sets of curves obtained from a large Rhesus monkey. The series of initial deflections shown to the left is that obtained after clamping the right division of the bundle. The central series comprises the natural curves obtained from the undamaged heart. The right-hand series shows the calculated dextrocardiograms. Abscissæ and ordinates as in Fig. 61.

For this reason it seemed expedient to examine monkeys. Between the monkey's dextrocardiogram (Fig. 67) and the supposed human dextrocardiogram there are no conspicuous differences, and a similar statement is applicable in the case of the levocardiogram.

* This feature is usually common to all three leads, but exceptions are found in lead *II* when the initial deflections are multiple or small.

† In some series of curves taken from the dog, the curves of lead *I* have different outlines, a fact which has led to misapprehension (*671*).

Considering the evidence as a whole, there can be no question that the curves described, and supposed to represent human levocardiogram and dextrocardiogram, are respectively due to defective conduction in the right and in the left divisions of the bundle.

Curves associated with preponderance of one or other ventricle.

It has been stated by Einthoven (*111*, *114*), that certain variations in the form of ventricular curves, as seen in the several leads, result from hypertrophy of the left or right ventricle. Einthoven appears to have based his view on the types of curve obtained in patients suffering from aortic disease and mitral stenosis. He described an exaggerated *R* in lead *I*, and an exaggerated *S* in lead *III*, as the effects of left hypertrophy (Fig. 68*b*) and the converse picture, namely an exaggeration of *S* in lead *I* and of *R* in lead *III* as the effects of right hypertrophy (Fig. 68*a*).

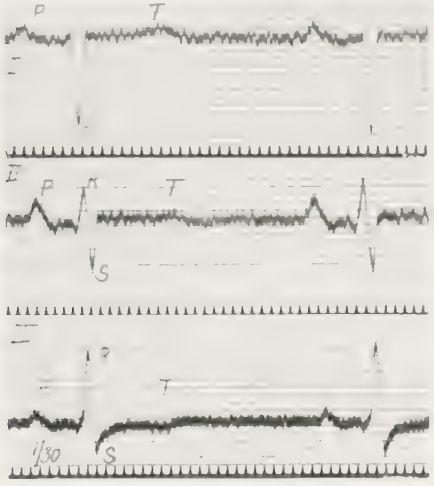


Fig. 68*a*.

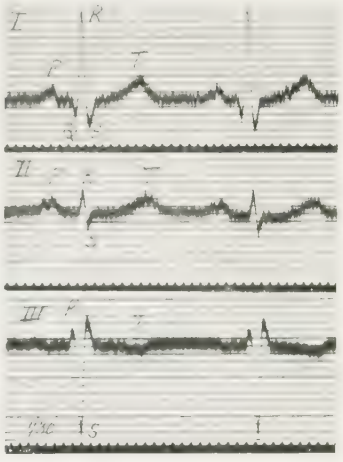


Fig. 68*b*.

Fig. 68*a*. Curves from the three leads in a patient in whom there was preponderating hypertrophy of the right ventricle. The chief deflection in lead *I* is *S*, in lead *III* it is *R*. Time-marker in thirtieths of a second.

Fig. 68*b*. Curves from the three leads in a patient in whom there was preponderating hypertrophy of the left ventricle. The chief deflection in lead *I* is *R* and in lead *III* it is *S*. Time in thirtieths of a second.

From time to time (*399*, *467*) further evidence in support of Einthoven's view has accumulated, and it is now sufficient to establish his signs as the most reliable which we possess of *preponderance* of one or other ventricle.

First, if a series of cases of mitral stenosis (or pulmonary stenosis) is examined electrocardiographically, the average curves obtained exhibit Einthoven's signs of right ventricular preponderance. The weights of the separated ventricles in a similar series shows in the average preponderance

of the right ventricle (*467*). Secondly, if a series of cases of aortic disease is examined, Einthoven's signs of left preponderance are found in the average curves, and the weights of the separated ventricles in a similar series show preponderance of the left ventricle.* Thirdly, the heart of the child possesses a relatively heavy right ventricle from the time of birth up till three months after birth. The signs of right preponderance are always present in the child at birth (Fig. 69) and disappear about the third month of extra-uterine life (*399*, *467*). Finally, in instances in which electrocardiograms have been obtained, and in which the weights of the separated ventricles have been taken, the correspondence between the electrical signs and the ratio of weights of right and left ventricles, has been remarkably exact (*467*).

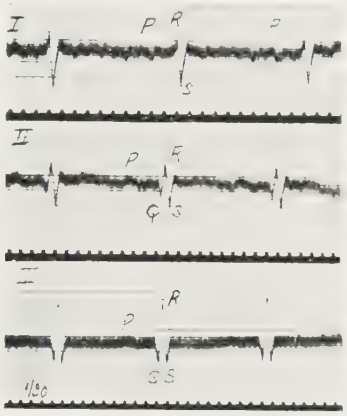


Fig. 69. Curves from a child two hours after birth. The relative heights and depths of the peaks is such as is expected where there is relative preponderance of the right ventricular muscle.

Einthoven's conclusions are confirmed by these observations, and there is little reason to doubt that they are valid.† The complete series of electrocardiographic signs, corresponding to preponderance of one or

* In some instances of mitral stenosis or aortic disease, preponderance, as estimated by weighing, is not discovered in the expected ventricle. Neither are Einthoven's signs of right hypertrophy discovered in all instances of mitral stenosis, nor those of left preponderance in all cases of aortic disease.

† A recent criticism (*42*) of my conclusions, in regard to the value of electrocardiograms in recognising preponderance of one or other ventricle, appears to be based on the assumption that if the electrocardiographic and clinical evidence seem incompatible, the electrocardiographic evidence must be at fault. Further misunderstanding appears to have occurred in that the distinction between the terms "hypertrophy" and "preponderance" is not fully grasped. I have endeavoured to make this distinction unmistakably clear. The signs described are not signs of enlargement of the heart, but of a disturbed balance between the mass of muscle on right and left side. The left ventricle may be hypertrophied and the signs of right preponderance appear if the right ventricle is hypertrophied in greater proportion. Moreover, the signs of one or other preponderance may be seen when the heart as a whole is not enlarged.

other ventricle, as they are now known to us (*475*) may be tabulated.

Right preponderance.		Left preponderance.	
Lead *I.*	Lead *III.*	Lead *I.*	Lead *III.*
Q R S	*Q R S*	*Q R S*	*Q R S*
- — +	+ + —	+ + —	— — +

Not only are there changes in the amplitude of *R* and *S*, but also in the amplitudes of *Q*; and these changes in right and left preponderance are qualitatively identical with the changes discovered when respectively the left and right divisions of the bundle are defective. Briefly, there is a close resemblance between the initial phases of the electrocardiogram when there is left preponderance and when the right division is defective; there is a similar resemblance between these phases when there is right preponderance and when the left division is defective. This observation has suggested the explanation of the curves associated with hypertrophy (*475*). To exemplify; if one ventricle (the left) preponderates, then in the dual curves taken from both ventricles, the levocardiogram will preponderate; and inasmuch as the left ventricle is the more massive, in so much will the levocardiogram impress itself upon the combined curve; in extreme instances of left preponderance, the electrocardiograms in their initial phases resemble levocardiograms in the closest detail (see Fig. 65*a* and *b*). Such curves, when analysed in respect of the rotation of the electrical axis, show a similar rotation to that observed in the case of the levocardiogram.

The initial phases of the levocardiogram, as opposed to those of the physiological curves, exceed 0·01 seconds in duration as has been stated; this increase is due to the defect in the right division of the bundle and to the consequent delay of spread in the right ventricle. A similar, though less pronounced, prolongation is seen in the curves of left hypertrophy, though here it is attributed to delay in the spread of the excitation wave consequent upon the thickness of the muscular wall of the left ventricle.

Usually, curves of hypertrophy may at once be distinguished from those of defects of the right division of the bundle, which they resemble, by comparing the initial phases of the curves. The characters of the levocardiogram are not fully displayed, and the increase in the duration of the initial phases is inconsiderable (compare Fig. 65*a* and 68*b*, and also Fig. 70 and 71). I have fortunately obtained (*465*) two series of curves showing the differences in a single patient (Fig. 70 and 71). They are striking and characteristic. Although the chief initial deflections in leads *I* and *III* are similar in direction in both series, the amplitudes of these deflections and their durations are much greater in Fig. 70. Moreover the direction of the chief deflection and the end deflection are opposite in leads *I* and *III* of Fig. 70; this is not the case in Fig. 71. The last distinction is one of considerable practical importance; for where (as in Fig. 65*b*) the initial characters of the levocardiogram are fully developed in curves which are, in reality, the result of left muscular preponderance, these curves are to be distinguished from curves expressing aberrant heart-beats by the amplitude and direction of the final deflection and by these criteria only.

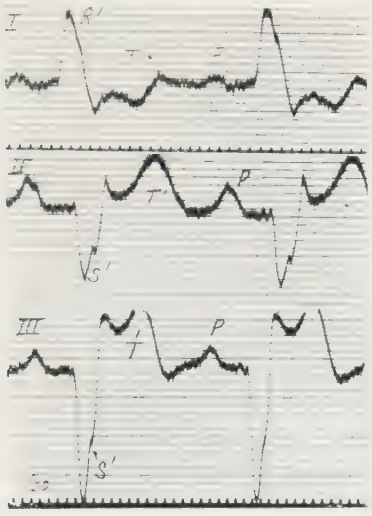


Fig. 70.

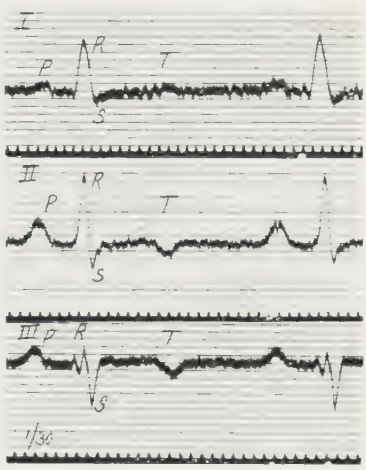


Fig. 71.

Fig. 70. Curves taken from the three leads in a case of aortic disease during a febrile attack. They show defective conduction along the right division of the auriculo-ventricular bundle.

Fig. 71. Curves from the same patient, taken a day later and during the subsidence of the fever. The ventricular portions of the curves have changed profoundly; there is now no evidence of bundle defect, but of relative preponderance of the left ventricle.

Minor forms of aberration.

Since it is known that the normal ventricular electrocardiogram is controlled in its form by the course which the excitation wave takes, and that the course of the wave is in turn governed by the arrangement of the *A-V* conducting system; and since it is known that a lesion completely transecting one whole bundle division profoundly modifies the form of curve, it is to be supposed on *a priori* grounds that lesser interferences with the channels which conduct the excitation wave in its descent to the ventricles will also leave clear impressions upon the resulting electrocardiogram.

This conjecture is completely upheld by experiment. I find in actual experiment that a lesion which interrupts any of the larger end branches of a bundle division will influence in greater or lesser degree the corresponding curve, according as it deflects the excitation wave in greater or lesser degree from its accustomed path. A lesion which cuts the right division of the bundle as it reaches the papillary muscles produces a conspicuously modified curve, though such a curve does not depart from the normal so greatly as does the curve resulting from a break in the division high up on the septum;* section of the chief end branches of the right division, where these are clear of the papillary muscle and bridge the cavity of the ventricle, produce lesser changes; lesions involving the right network as it lies beneath the endocardium do not greatly change the curves. Similar conclusions apply to the arborisation in the left ventricle.

* Hence it is to be recognised that there are early outgoing branches from this bundle division.

It is certain that lesions in the lower reaches of the arborisations occur in the human subject; there is every ground for the belief that such pathological damage modifies the human electrocardiogram more or less in the corresponding patients.

To identify lesions in particular regions of the arborisation with corresponding forms of electrocardiogram is a task which lies in the future.

But there is another way in which the electrocardiogram may suffer change in form, namely, by variations in the relative *rates* of conduction along the two divisions of the bundle and their branches. When the left division is so nipped in the jaws of a clamp that conduction through this tract is abolished, the curve in its initial phases is that of the right ventricle only (the dextrocardiogram). If the clamp is now relaxed, the power of the division to conduct will frequently return. But the recovery takes time, and during this stage of recovery a form of electrocardiogram is seen which is often transitional between the normal curve and the dextrocardiogram. Clearly, since the actual form of the initial deflections is due to a summation of dextrocardiogram and levocardiogram, the form of these deflections and their amplitudes will depend largely upon the times of events in right and left ventricle in relation to each other. A delay in the appearance of levocardiogram or dextrocardiogram and consequent distortion of the bicardiogram is strongly suspected to occur in certain patients. Some years ago I advanced this hypothesis of delay in the recovery of conduction in a single bundle division to explain a striking and remarkable series of transitions between a complex having the full features of a levocardiogram and a normal complex (*451*). The changes occurred over a series of eight ventricular cycles, each ventricular complex being intermediate in type between those adjacent to it. This curve (Fig. 29 of my paper) was taken from an asphyxiated cat in whose auriculo-ventricular system conduction changes were known to be occurring. Clinical examples of an almost parallel kind have been published by Cohn (*61*) and are probably to be explained similarly. Many other clinical examples of temporary disturbance of a bundle division have since been recorded (*182*, *543*, *581*, *783*). Without exception, so far as I am aware, curves showing the gradually changing complexes (from dextrocardiogram to levocardiogram, from levocardiogram to dextrocardiogram, or from one or other to a curve of the normal type), such as are here described, have been obtained from patients in whom there were unequivocal signs of deficient conduction in the main bundle, or strong evidence of its presence.*

* In Frederica and Möller's case (*182*) of auricular fibrillation, I judge complete heart-block to have been present, although the authors seem not to have been of this opinion. Their curves show the gradual development or gradual disappearance of the dextrocardiogram. At autopsy a lesion of the septum, involving the left bundle division was discovered. The curves are not those of right preponderance as the authors would suppose, for the initial phases are prolonged and the direction of *T* is opposite to that of the chief deflection in all leads of the curve.

Note.—Another mechanism by which a gradual transition of curves from one type to another is produced, is described at page 217.

Displacement of the heart's anatomical axis.

In Chapter VIII methods of calculating the heart's electrical axis have been considered, and it has been seen that this axis varies very greatly during the progress of the cardiac cycle. It is to be emphasised again that none of these electrical axes can be assumed to represent the anatomical axis. But as it is certain that the electrical axis bears to the anatomical axis a certain relation, inconstant though it may be, it is true that the former may be employed within certain limits in calculating the actual lie of the heart in the body. There is no better example to illustrate this fact than the transposed heart; for, as in most normally placed hearts the direction of the electrical axis corresponding to the phase when *R* is inscribed is inclined to the horizontal at approximately 75°, so in transposed hearts it is usually inclined at approximately 105°, being as much inclined (by 15°) to the right of the vertical in the former, as to the left of the vertical in the latter. But inasmuch as there are other factors which influence the direction of the axis when *R* is inscribed, such as a relatively greater development of one ventricle or the other and individual differences in the distribution of the excitation wave within the ventricles, in so much is this method of ascertaining the relative position of the anatomical axis in different individuals uncertain.* Nevertheless, in calculating changes in the direction of the anatomical axis in one and the same individual, changes such as may be induced by posture, breathing or other act which displaces the heart, the method is not unserviceable. Conversely, a chief merit of the method is that it explains many of those changes in the form of the electrocardiogram which are seen to result when the lie of the heart becomes altered. To simplify my description of these changes of form I have constructed the accompanying diagram (Fig. 72).

If we suppose a manifest potential difference of fixed value to be developed in the chest and imagine further that it rotates through a full circle in the plane of the leads, then this potential difference, as it rotates from 0° to 180° and again through to 0° will be represented in the three leads in the fashion shown in the diagram. Thus, in lead *I* when the axis lies at 0° (see left hand edge of e^1 in Fig. 72), and therefore in the line of the horizontal lead, the difference in potential will be fully represented and will be shown as a full upward deflection. As the axis rotates from 0° to 90° the difference of potential shown by lead *I* will gradually decline until it reaches zero, for at 90° the axis is at right angles to the lead. On further rotation in the same clockwise direction, the potential difference will again gradually increase, though the direction of the deflection will now be reversed (downwards) until at 180° it will again reach its maximum, for again the axis and the lead will be in the same line. During

* That there is a relation between the type of electrocardiogram and the lie of the heart, estimated orthodiagraphically or skiagraphically, is stated (*233, 747*), and no doubt this relation exists; but the lie of the heart will not account for many conspicuous differences in individual curves taken from healthy people.

the further rotation from 180° to —90°, there will be a second gradual decline to zero, and during the last part of the rotation, from —90° to 0° there will be a gradual increase until the original upward maximum is reached. Similar changes will occur in leads *II* and *III*, though at different phases of the rotation, as shown. This diagram is so constructed that the relative values of e^1, e^2 and e^3 are accurately shown for all positions of the electrical axis in its revolution. Thus, when the axis is 60° the relative values of e^1, e^2 and e^3 are 5, 10 and 5 respectively, where 10 is the value of the manifest potential E (see Fig. 58*a*, page 101).

Now in the most usual form of normal electrocardiogram the angle which the axis, while *R* is inscribed, makes with the horizontal averages approximately 75°. If the heart is tilted so that its anatomical axis moves in an anti-clockwise direction through some 30°, the electrical axis will follow through 30° and will move from 75° to 45°. During this movement (see Fig. 72) *R* in lead *I* may be expected to increase considerably from its original small value, in lead *II* it will at first increase a little and later decline a little, while in lead *III* it will steadily and conspicuously decline. The chief change, be it observed, will occur in leads *I* and *III*, and the changes will be in opposite directions. These are precisely the changes which are seen to occur in the height of *R* in these leads, when the apex of the heart is tilted upwards and to the left, as when gas is introduced into the stomach (*323*), or when the diaphragm rises in expiration.

In the same type of electrocardiogram the angle of the axis while *T* is inscribed, averages approximately 55°. If the heart is tilted through 30°, this angle will be decreased from 55° to 25°. In lead *I*, *T* should increase conspicuously in size, in lead *II* it should show some decline in size, while in lead *III* it should diminish to zero and even become slightly negative. These also are precisely the usual changes seen in *T* in full expiration or when the stomach is distended. The axis while *S* of the usual electrocardiogram is inscribed is very variable, and the changes which have been observed in its magnitude during the phases of respiration are also variable. The data which we possess are insufficient to formulate any precise rules. Writers have for the most part not taken into account the original angle while *S* is inscribed, and to this the lack of constancy in the changes of *S* is very probably in large part attributable.*

In a less usual type of electrocardiogram *R* is originally as high or almost as high in lead *I* as in lead *II*, while it is inconspicuous in lead *III*. The axis corresponding to it lies in the neighbourhood of 35° (see Fig. 72). In such cases, anti-clockwise rotation of the heart leads to a slight increase of *R* in lead *I*, a steep decline in lead *II*, and inversion in lead *III*. These are again precisely the changes to be anticipated.

* Lack of constancy, so it is thought, may also result because rotation of the heart on its vertical axis is thought to be particularly prone to affect *S*.

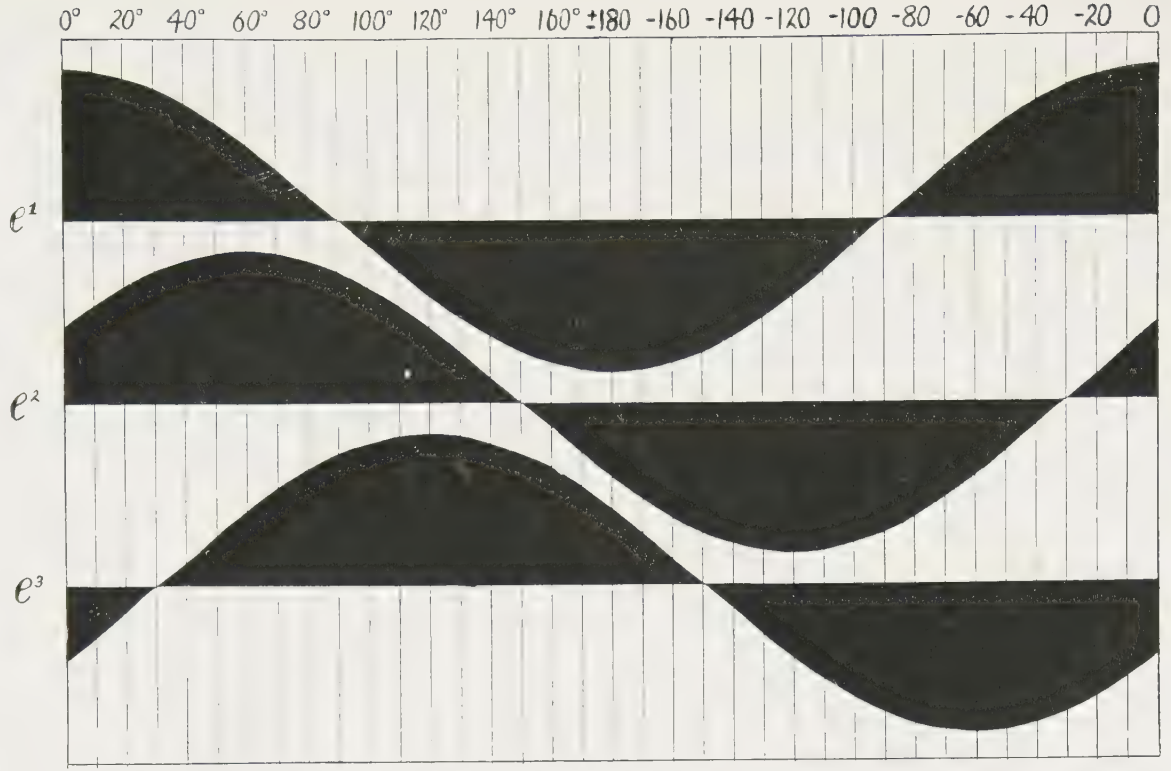


Fig. 72. A diagram of the differences in electrical potential developed between the contacts in leads *I*, *II* and *III* (e^1, e^2 and e^3) when a manifest potential of constant value is revolved through 360°. The angles formed by the electrical axis are represented by the abscissæ and are marked above the chart. The reading gives the inclination of the axis to the horizontal and starts at 0°, when the axis is horizontal and in line with lead *I*. The axis is supposed to rotate in a clockwise fashion until at 180° it again comes into line with the horizontal: from this point the axis, rotating still in a clockwise fashion, passes back through 180° until it takes up its first position.

The diagram may be found serviceable in quickly gauging in an approximate fashion the angle of the electrical axis, when the relative values of the corresponding deflections in the three leads are known. The diagram may be used in this manner for any deflection of the electrocardiogram.

It should be observed that in considering the changes which occur in the heights and directions of electrocardiographic deflections in response to tilting of the heart, the original directions of the various axes have clearly a very important influence.

I have described somewhat fully the changes seen in an anti-clockwise rotation; the changes in a clockwise rotation, such as deep inspiration will produce, are of the reverse order.

Changes in the form of the electrocardiograph during respiration were first noticed by Samojloff (*679*), and were later studied by Einthoven (*114*, *116*, *119*), Grau (*233*), Kahn (*358*, *359*), and others (*323*). It is to Einthoven and his associates that we chiefly owe the explanation of these changes. The descriptions are not always, though they are in the main, uniform; the lack of uniformity is largely attributable to the different type of electrocardiogram dealt with. In the case of expiration and inspiration there are, however, other factors which come into play for, accompanying the acts of breathing, the vagal tone alters* and the shapes of the curves are thereby influenced; some change is also induced in all probability by rotation of the heart around its own axis (*114*).

USUAL CHANGES IN NORMAL ELECTROCARDIOGRAMS WITH RESPIRATION.

P	*R*	*S*	*T*		
+	—	+ —	—	lead *I*	inspiration.
+	+	— +	+	lead *III*	
—	+	— +	+	lead *I*	expiration.
—	—	+ —	- - (Neg.)	lead *III*	

Displacement by pleural effusion does not appear materially to influence the electrocardiogram (*323*), for the withdrawal of a large quantity of fluid from one side is usually unaccompanied by any great changes in the curves.† The absence of much change is probably to be explained by the displacement of the heart occurring as a whole and without much rotation of its axis.

The chief change which has been observed to result from change of posture is a conspicuous deepening of *S* in lead *I*‡, when the subject rolls from the left to the right side. Einthoven believes this change to be due to a rotation of the heart upon its own axis, for it is not usually accompanied by any material change in *R* or *T*.

* As recently emphasised by Blumenfeldt and Putzig (*35*). The conclusions of these writers apply, however, to the dog, in which vagal tone changes vary greatly with respiration; they cannot be applied without reserve to the human electrocardiogram. The changes in *P* in the human curves are probably largely controlled by the vagus (see page 366).

† The changes which accompany displacement of the heart when an inert gas is introduced into one pleura in quantities sufficient to produce collapse of one lung are, in my own experience, trifling.

‡ There may be also an increase of *T* in this lead (*233*).

Chapter X.

The Analysis of Disordered Mechanism.

*Arterial Pulse Curves.**

Arterial pulse beats indicate contraction of the left ventricle, but they may be utilised in studying disorders of the cardiac mechanism upon a broader basis ; for as we have no evidence of the isolated contraction of right or left ventricle, they may be taken to signal contraction of the ventricular musculature as a whole.

Arterial curves in which there is variation from beat to beat, fall into two broad classes : (1) those in which the pulse rhythm is regular, but in which the individual beats vary in amplitude (for example, respiratory variations of excursion, and the pulsus alternans, Fig. 73) ; (2) those in which the pulse rhythm is irregular.

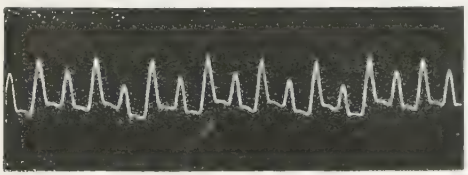


Fig. 73. An example of irregularity in the excursion of radial pulse beats. The sequence is regular, but alternate beats are of large and small excursion. A condition known as "pulsus alternans" (see Chapter XXXIII).

It is the pulse showing irregularity in the incidence of its beats which is now our special concern. A close study of irregular arteriograms is very profitable ; the type of disordered heart action may usually be identified from the pulse tracing alone. In polygraphic records, the arterial tracing should always command first attention ; most false interpretations result from its neglect. But although the arterial pulse serves us in this manner, it should not be forgotten that this method of analysis has come only after extensive observation ; and that, while it often suffices to give a clinical diagnosis, *proof* of a particular disorder often, nay usually, requires polygraphic or electrocardiographic confirmation.

* The reader who is unacquainted with interpretations of disordered heart action may to advantage delay reading this chapter and the two which succeed it, or pass over them quickly, until he is more familiar with the types of disordered action seen clinically.

It will not be possible in the present chapter to review the whole of the information which may be reaped from arterial curves, and I shall be content in exemplifying the principle features of the method. Pulse irregularities may be divided into a few simple categories; some are of simple form, others are extremely complex.

Pulse intermissions.

The simplest form of irregularity is that in which the pulse "intermits"; the regularity of the pulsations is interrupted at occasional or frequent intervals by a pause of unusual length. Disturbances of this kind will serve as preliminary illustrations. In the top tracing of Fig. 74, a regular pulse is disturbed at a single point, a long cycle *b* is seen, and the disturbance is of such a kind that the intervals *a* and *b* are equal. A single beat seems to be missing from the curve; otherwise it is unchanged, for the rhythmic beats which follow fall accurately at expected points; they are in their proper places, the sequence being a direct continuation of the old sequence. The heart's action is controlled throughout by a dominant rhythm, which continues over the period of interruption and is not itself disturbed.

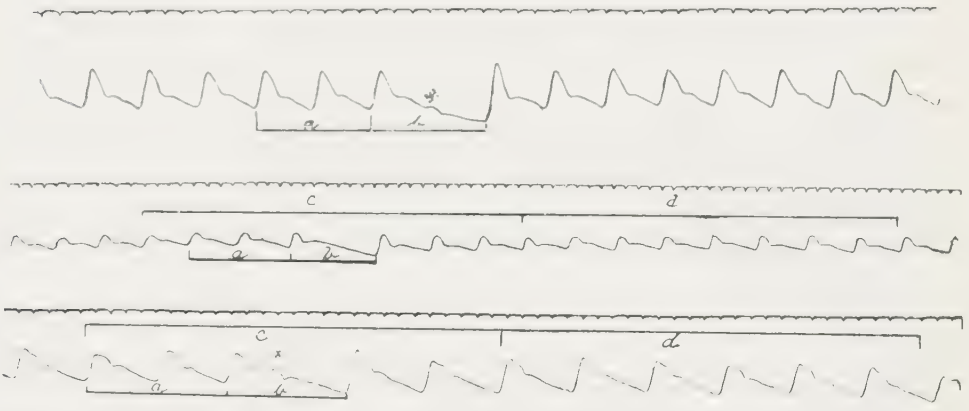


Fig. 74. (× ⅔.) Three radial pulse tracings from patients, showing solitary intermissions. In the top tracing the irregularity results from a single premature and weak contraction of ventricular origin; the weak beat shows in the tracing and is marked by an asterisk. The disturbance is of such a kind that the intervals *a* and *b* are equal. The dominant or controlling rhythm of the heart is undisturbed. The middle tracing shows an intermission resulting from heart-block; interval *a* is longer than interval *b*; yet the dominant rhythm is undisturbed, as is shown by measurement over the whole period of disturbance; intervals *c* and *d* are equal. The bottom tracing shows an intermission resulting from a premature and weak contraction of auricular origin; interval *a* is longer than interval *b* and measurement of the disturbed stretch of tracing (interval *c*) against an equal stretch of undisturbed tracing (interval *d*) informs us that the dominant rhythm has been disturbed at its source; the contractions following the disturbance do not occur at expected points.

In the middle tracing of Fig. 74 the pulse is also interrupted by an unusually long cycle ; but, in contrast to the events of the top tracing, the interval *b* is shorter than the interval *a*. Yet when we measure the whole period of the disturbance, taking in a few beats before and a few beats after the pause, we find that this interval (*c*) corresponds exactly to that covering a number of regular pulse beats in the same tracing (interval *d*). As in the top curve, we have to deal with an irregularity which does not interfere with the dominant rhythm, for the pulse beats are eventually restored to their natural positions. But in the present instance we deal with a less simple form of irregularity ; not only is there an unusually long pulse cycle, but the lengths of the pulse cycles immediately before and immediately after the disturbance are changed. A disturbance of this kind always results from heart-block.

Finally, in the bottom tracing of Fig. 74, we see a third variety of intermission ; as in the middle tracing the interval *b* is shorter than the interval *a*, but in addition to this irregularity, the dominant rhythm is disturbed, as is clearly shown by measurement over the whole period of disturbance (bracket *c* = bracket *d*). A disturbance of this kind is the result of a premature contraction arising in the same heart chamber as the dominant rhythm ; in this instance it arose in the auricle.

The dominant rhythm may be defined, therefore, as a rhythm which governs, more or less, the ventricular movements. It is a rhythm which, in a case of irregularity, is disturbed at its source, or which has its impulses disturbed during transmission from their source to the ventricle ; usually the dominant rhythm arises in the pace-maker, and a very large number of ventricular irregularities depend upon disturbance of the natural and regular flow of impulses from this point to the several cardiac chambers. So far as the study of cardiac irregularities has proceeded, the rhythm dominating the ventricle, in any given case, may be asserted to be the one which is generated at the highest level of that portion of the cardiac tube from which impulses pass to the ventricle. This is usually the pace-maker, but occasionally other points may assume control.

Where a dominant rhythm is present, those pulse beats which are preceded by the longest pulse cycles are usually initiated by it ; and in long stretches of curve such beats are most conspicuous in the tracing by reason of their amplitude. In searching for a dominant rhythm, these large beats command our chief attention.

Signs of a dominant rhythm.

The first and most essential step in the analysis of irregular arteriograms consists in trying to establish the presence or absence of a dominant rhythm. The curves of Fig. 74 present no difficulty in this respect, for over the greater part of each the beats are placed regularly. Whenever runs of perfectly regular beats occur in tracings which are otherwise irregular, and these beats

are the most forcible seen, such beats belong to the dominant rhythm. A run of four or more such beats, occurring from time to time, always reveals a controlling influence of this kind.

If the pulse beats appear in pairs or in groups of three four or more pulsations, the presence of a dominant rhythm is certain, providing that the individual groups resemble each other in all respects. If in the case of very irregular pulses, the time intervals between the largest beats fall into a relatively few categories, the same is true; a dominant rhythm is also displayed if the phases of irregularity are repeated, however irregular such phases may be. (Fig. 75 and 76). In brief, the presence of a controlling

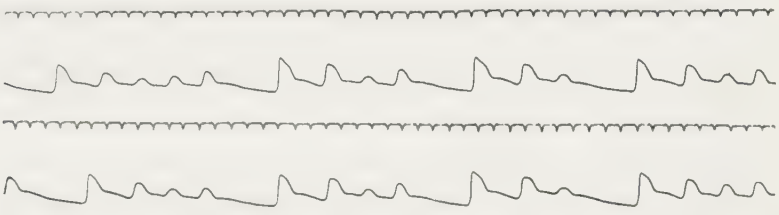


Fig. 75. Two portions of a long arterial curve, showing repetition of the same phase of irregularity and providing conclusive evidence that the irregularity is controlled. The curves were taken from a case of flutter.

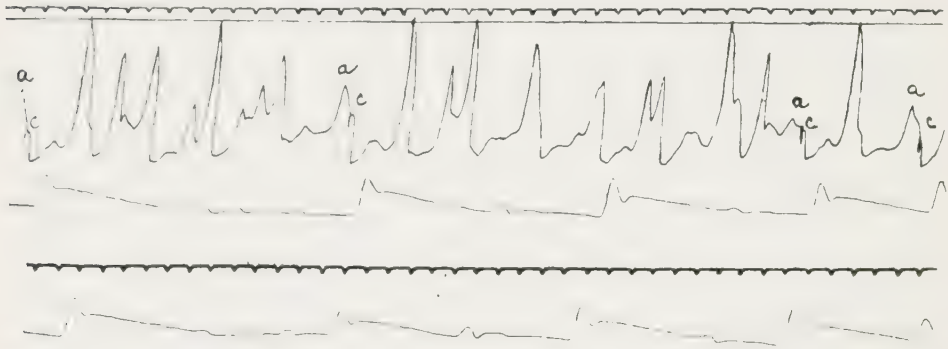


Fig. 76. ($\times \frac{2}{3}$.) Two radial curves from a single patient. The upper one is accompanied by a venous curve (the two venous curves were identical in every respect, the lower one is therefore omitted). To illustrate the presence of a dominant rhythm; most of the prominent radial beats belong to it. The lengths of the intervals between prominent beats fall into a few categories. A period of irregularity of moderate length is duplicated. The irregularity resulted from premature contractions arising in the ventricle (the actual events were determined electrocardiographically).

rhythm may be assumed whenever an irregular pulse shows periodicity of any kind. These facts are of special importance in excluding irregularities of the ventricle which are due to fibrillation of the auricles (see Chapter XXIII.).

Signs of an undisturbed dominant rhythm.

After ascertaining that a dominant rhythm is present in a given case, it is often possible to carry the analysis a step further, and, as we have seen, to show that the dominant rhythm is unaffected by the ventricular irregularity (Fig. 74, top curves). If a pulse in which the beats are paired or grouped occasionally becomes regular and there is a simple mathematical relation between the intervals separating groups and the intervals separating regular beats (Fig. 77, top curves), the same dominant rhythm controls each portion of the curve, and this rhythm is unaffected by the irregularity. An example, in which the controlling rhythm is affected, is shown in the bottom curve of the same figure. The three curves of this figure are more complex instances of what we have seen in Fig. 74.

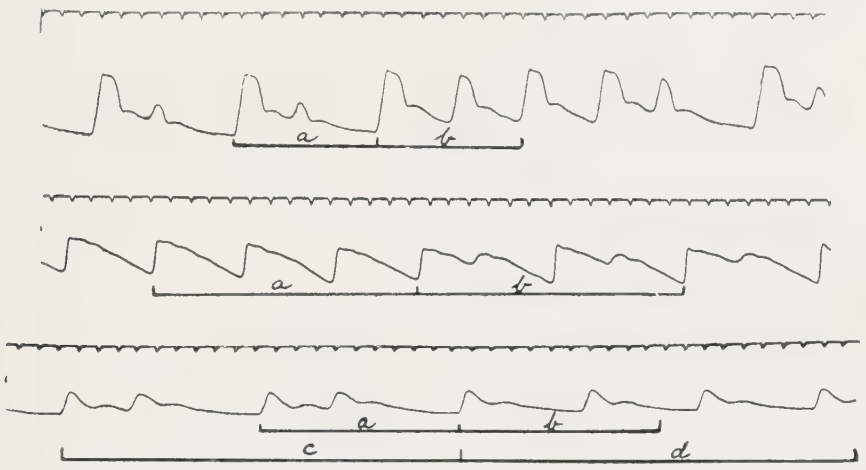


Fig. 77. Three arterial curves from patients showing coupled pulse beats and transitions to regular heart action. In the upper two curves there is a simple mathematical relation between the lengths of the paired beats and the lengths of the regular beats. The same dominant rhythm controls the arterial curve, whether its beats are in pairs or are regular, and this rhythm is undisturbed. In each of these curves the intervals *a* and *b* are equal. In the bottom curve this relation between the paired and unpaired beats is not found; the presence of a dominant rhythm is known because the two groups of paired beats are equal to each other in length, and because the three succeeding beats are equal to each other in length; but the measured intervals *a* and *b* and *c* and *d* show that this dominant rhythm has been disturbed. The actual events in these and succeeding tracings were determined polygraphically and electrocardiographically.

Further, when the pulse beats occur in uneven groups and simple mathematical relations may be established between the lengths of all the groups, the same conclusion holds good, even if the individual groups have not the same detailed construction (Fig. 81). The stability of a controlling rhythm may be demonstrated in irregular curves of very great complexity; examples are shown in Fig. 78-80.

The same principle is applied to curves in which there are abrupt changes of rate, the heart beating regularly before and after the change; for if there is a simple relation between the slow and fast periods, the fundamental rhythm of the heart has remained unaltered over the change; while, if no such relation can be established, the faster rhythm is a new development and interrupts and supersedes the fundamental rhythm (Fig. 82).

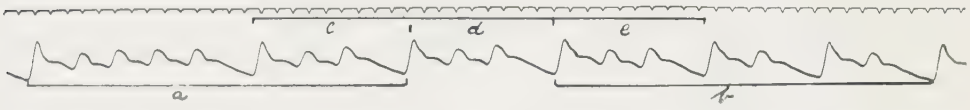


Fig. 78. ($\times \frac{2}{3}$.) An arterial curve from a case of flutter, showing an undisturbed dominant rhythm. The stretches of curve *a* and *b* are equal; and these two stretches are dovetailed by three equal groups (*c*, *d* and *e*)

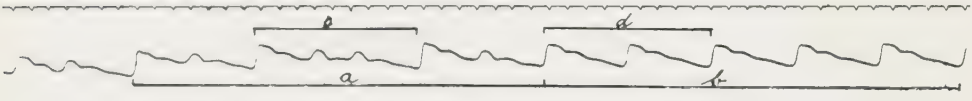


Fig. 79. ($\times \frac{2}{3}$.) An arterial curve from a case of flutter showing two equal stretches of curve one of which (*a*) includes two groups of paired beats and a group of three beats, the other consisting of five regular beats. The periods *c* and *d* are also equal. The dominant rhythm is undisturbed

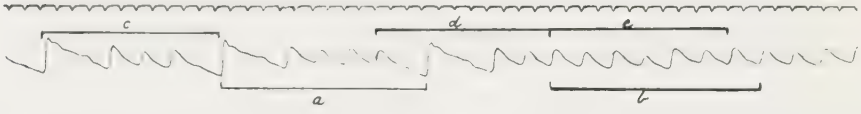


Fig. 80. ($\times \frac{2}{3}$.) An arterial curve from a case of flutter. The stretches *a* and *b* are equal; the stretches *c*, *d* and *e* are also equal. The whole curve is covered by measured stretches which adjoin or overlap. The whole curve shows evidence of the same and undisturbed dominant rhythm. It should be noticed especially that measurements of this kind are only justified when the beats, lying at the extremities of any given bracket, have equal pauses preceding them.

In Fig. 78, 79 and 80 the irregularities resulted from heart-block.

When the dominant rhythm is undisturbed, a pulse irregularity is due to heart-block or premature ventricular contractions.

A regular sequence of beats originates from a single source.

When a pulse is regular for long periods it may be taken that the rhythm is promoted by impulses arising in a small and limited area of the musculature, and probably from a single point. And this statement holds true whether the rhythm originates in the normal pace-maker or not.

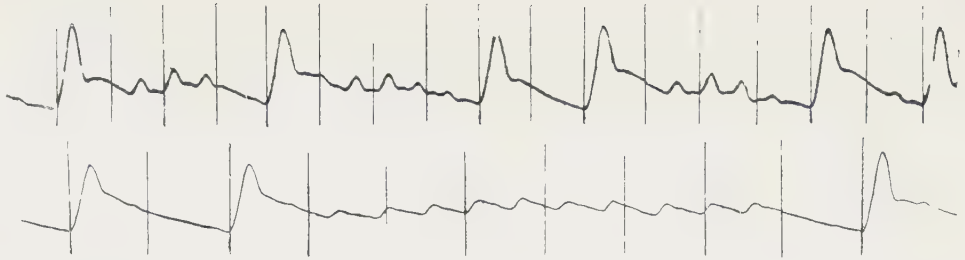


Fig. 81. Two arterial curves from a patient, showing great irregularity of the ventricular movements, without disturbance of the dominant (in this case, auricular) rhythm. The large beats in each curve fall exactly upon a series of vertical lines placed at regular intervals. The irregularities resulted from premature ventricular contractions.

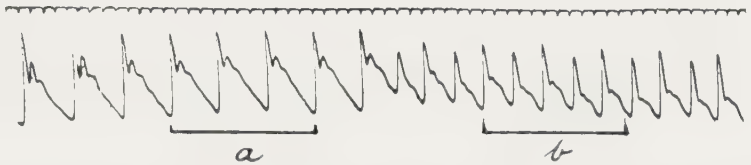


Fig. 82. A radial curve from a patient showing an abrupt change of heart rate. As there is no simple relation between the rates of the two regular rhythms to right and left, the faster is not under the control of the original dominant rhythm. It is due to the interference of a new or pathological rhythm, a paroxysm of tachycardia (see Chapter XX).

Fig. 83. (× ½.) An example of phasic variation of a dominant rhythm from a lad, the variation is independent of respiration. All impulses arose at the pace-maker (as shown electrocardiographically). The curve shows the characteristic and *gradual* passage from slow to fast rates. Intervals of varying lengths are arranged in orderly fashion in relation to each other.

Observation permits the further statement that, where a number of pulse beats are found, for example six or more, and they are equal in amplitude and regular in their succession, the impulses from which they originate are generated in a single focus, whether the rhythm of which they are an expression is dominant or interrupting. An example of an interrupting rhythm of 10 beats arising from a single point in the ventricle is shown in Fig. 81.

The method of examining an arterial curve now described I call the "spacing" of curves.

Phasic variation of the dominant rhythm.

The reverse statement, that a rhythm generated at a single focus is regular, is not necessarily true. For if such a rhythm is built up at the pace-maker it frequently shows periodic variations of rate. It is often possible to identify these variations in the arteriogram. They consist of a gradual waxing and waning of pulse rate, which is repeated and is usually synchronous with the acts of breathing. Phasic variations of rhythm which are independent of respiration also occur (Fig. 83), but cannot be identified certainly without the aid of records from other heart chambers. (Variations of this character are more especially considered in Chapter XXXII.)

The rate of the dominant rhythm as an index of its source.

The rate of the dominant rhythm, usually to be ascertained in the arteriogram, offers a clue to the source of such a rhythm, though it is not decisive in this respect. The dominant rhythm may have its origin in the normal pace-maker, and under these circumstances the rate usually approaches 72 to the minute; but the limits of variation are great and pass from 30 to 240 to the minute. The dominant rhythm may have its origin in the ventricle, and under these circumstances its rate approaches 30 to the minute (the known limits in man are from less than 1 to 90 to the minute). Not infrequently a new rhythm of pathological type may prove dominant, and such rhythms may arise at many points in the musculature. The rate of these new rhythms is variable and the limits approach 110 and 343 per minute.

Thus if the dominant rhythm has a rate approaching 70, there is presumptive evidence that it is generated in the pace-maker. If the rate is approximately 30, the rhythm is usually of ventricular origin. Lastly, if the rate lies constantly and continuously between 130 and 340 new or pathological impulse formation should be suspected.

The length of the cycle following a premature beat.

This measurement is of value in indicating the source of the premature contraction and will be referred to in more detail at a later stage. At present the following categorical statements may be made in regard to such cycles:—

1. If the cycle is of a length equal to that of a cycle of the dominant rhythm the source of the premature beat and the source of the dominant rhythm are the same.

2. Further, it may be said that the greater the distance between the points at which dominant rhythm and premature contraction arise, the longer will be the cycle following the premature beat.

3. And finally it may be stated that if the cycle following the premature beat is of full length, so that it compensates for the curtailment of the previous cycle re-establishing the spacing of the dominant rhythm, in all probability the premature contraction has arisen in the ventricle* (Fig. 74).

Complete irregularity of the pulse.

When a pulse is completely irregular, that is to say when the intervals between beats are of very varying lengths, when there is no regular lengthening from beat to beat or subsequent regular shortening from beat to beat, when phases of irregularity are not repeated periodically, when the lengths of beats do not fall into a few simple categories, a dominant rhythm is absent, and the heart's mechanism is one known as fibrillation of the auricle (Fig. 84).

The value of the arteriogram to the student of disordered mechanism can hardly be exaggerated. The early observations of Cushny (*86*), and especially of Wenckebach (*754-758*), in which arterial curves were critically studied, laid the basis of our present knowledge. Wenckebach's insight led him to differentiate a large number of the more important disturbances in these curves alone. My electrocardiographic studies have enabled me to extend his method (*447*, *455*, *457*).

Fig. 84. ($\times \frac{2}{3}$.) Complete irregularity of the pulse. From a patient with fibrillation of the auricle. The intervals from pulse beat to pulse beat show the wide variation characteristic of the condition. The lengths of the beats are marked in fifths of a second below the curve.

* Premature contractions arising in the auricle and junctional tissues occasionally give rise to this picture, but these instances are exceptional.

CHAPTER XI.

THE ANALYSIS OF DISORDERED MECHANISM.

VENOUS CURVES.

THE arterial pulse signals the ventricular systole; the venous pulse signals the auricular systole: these are our chief objectives in using the arteriogram and the phlebogram, respectively. The simultaneous or polygraphic record, indicates the time relations of these systoles to each other, and displays the sequence or lack of sequence in chamber contraction. The polygraph was the first instrument employed for this purpose; it is to Mackenzie (*499, 500*), that we chiefly owe a method which still maintains its lead as a routine device for clinical purposes.

Identifying the "c" wave.

Generally speaking the radial curve is taken as the standard from which all measurements are made, for the artery at the wrist presents natural advantages, rendering it the most serviceable point of arterial pulsation. The radial upstroke is used in calculating the onset of ventricular systole, and it is first corrected from a simultaneous carotid curve in order to allow for the difference in transmission time from heart to radial and heart to carotid, respectively. In this way a point is obtained upon the radial curve a little (about 0·1 sec.) before the upstroke, which represents the time at which the upstroke of the carotid occurs in the neck. In simultaneous venous and radial curves this point is transferred to the venous curve, and *in the latter* it represents the onset of ventricular systole. (These measurements may be made in Fig. 85.) Briefly, the measurements identify the upstroke of the *c* wave. The two measurements may be accomplished by a single transference. A short strip of venous and radial curves is taken; the clock is stopped, and index marks are written; short strips of carotid and radial curves are then obtained. The interval (*r r*) between two radial upstrokes, one lying to the left (Fig. 85) the other to the right of the index marks is measured. A corresponding carotid upstroke to the right of the stops is determined, and also a point upon the venous curve, separated from the carotid rise by the distance (*r r*) ascertained in the previous measurement. The transferred measurement is justified when the clock is running at the

same rate both to the right and left of the index marks, and when the curves to right and left are level, and only under these conditions. The question of level has always to be considered in using levers which describe an arc.* Where accurately measured intervals are desired, correction from the actual index marks is essential, and the points obtained should be transferred to the time marks. Where the waves of the jugular curve are simple in form and clearly inscribed, a single measurement is taken from the index mark to the upstroke of a radial beat to the left of it, and is transferred from the upper index mark to a point on the venous curve ; the upstroke of the *c* wave will

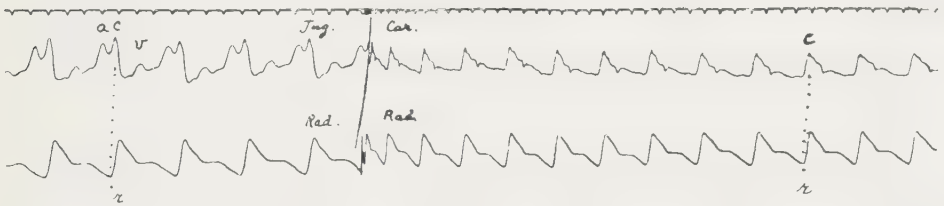


Fig. 85. ($\times \frac{2}{3}$.) Polygraphic curve, illustrating a method of identifying the *c* waves. Simultaneous radial and jugular curves are shown to the left, and simultaneous carotid and radial curves to the right of the central index marks. The transferred measurement is from *r* to *r* in the radial curve and from *c* to *c* in the upper tracings.

lie at approximately 0·1 sec. to the left of the point thus ascertained. The wave *c* is used as an *index* of the onset of systole ; the *actual* instant of onset may be obtained from a simultaneous cardiogram, mechanical or electric, or may be gauged approximately in a venous curve by allowing for the transmission intervals from heart to aorta (presphygmic interval) plus aorta to carotid ; a total allowance of about 0·1 sec. to the left of the carotid upstroke.

Identifying the "a" wave.

In a venous curve, where a group of clearly inscribed waves accompanies each cardiac cycle and where one of these waves lies directly before (to the left of) the upstroke of *c*, this wave is known to be *a*, and its upstroke represents the onset of the auricular systole in the venous curve. Its onset can be ascertained with certainty when the wave is prominent and clean cut from start to finish. Where the wave shows division, or where the upstroke lies at more than 0·2 sec. from the onset of *c*, the curve should be lettered with greater caution. Constancy in the shape and position of the wave from cycle to cycle or from one phase to a similar phase of the same curve is most

* No such correction is needed when the curves are taken photographically, as in Fig 28, page 51.

desirable; it is a golden rule only to accept those curves of which the interpretation is transparent, or of which repetitions in detail are secured. Where the limits of *a* are dubious, they should be checked from *a* waves at other points in the neck, from similar waves upon the apex curve, or lastly from evidences of auricular systole in electrocardiograms.

The "As-Vs" interval.

The interval *a-c*, measured between the commencements of the corresponding waves and taken as an *index* of the *As-Vs interval* (the true interval separating the commencements of auricular and ventricular systole), is customarily 0·1 to 0·2 sec.. It is usually longer than the corresponding *As-Vs* interval as shown by electrocardiograms, probably because *c* is more delayed than *a*. But this does not appreciably detract from the value of the *a-c* interval as an index.

The *a-c* and *P-R* intervals never differ from each other by more than 0·1 sec. (generally the difference is less), and consequently a prolongation of the *a-c* interval to 0·3 sec. or more is a reliable guide to similar lengthening of the *As-Vs* interval. Even greater reliance may be placed upon a notable change in *a-c* interval from beat to beat in one and the same case.

The value of the summit "v."

The upstroke of *c* is determined and checked from the arteriogram. The onset of *a* cannot be checked in a similar manner, therefore the measurement of *a-c* intervals should be circumspect. To determine the limits of ventricular systole is important in cases where *v* may be mistaken for *a*, or in instances in which *a* and *v* are suspected to coincide (in cases where the heart beat is rapid and *v* is insignificant, or in cases where the *a-c* interval is prolonged).

The end of ventricular systole is represented in sensitive carotid and radial curves by the dicrotic dip, and in the jugular curve by the apex of *v*. The summit of the latter is synchronous with a point lying a little (usually 0·1 sec.) after the bottom of the dicrotic depression in the carotid. The apex of *v* in the jugular is synchronous with the depression of the dicrotic in the radial curve. The summit of *v* may be also checked by measuring the length of the systole in a single carotid beat and transferring it to the jugular curve. The measurement is of practical value because the summit has so constant a relation to the end of systole; a relation which is well shown in Fig. 28, page 51. An example of its application may be studied in Figs. 91-94.*

* Theoretically the use of the dicrotic notch in this fashion in polygraphic work is open to criticism, for in crude curves the dicrotic dip is not properly represented; the actual dip which is seen is often largely an artifact. In practice, however, only a rough gauge is usually required, and experience proves these measurements to be sufficiently exact. They are of special value when used for different cardiac cycles in one and the same tracing

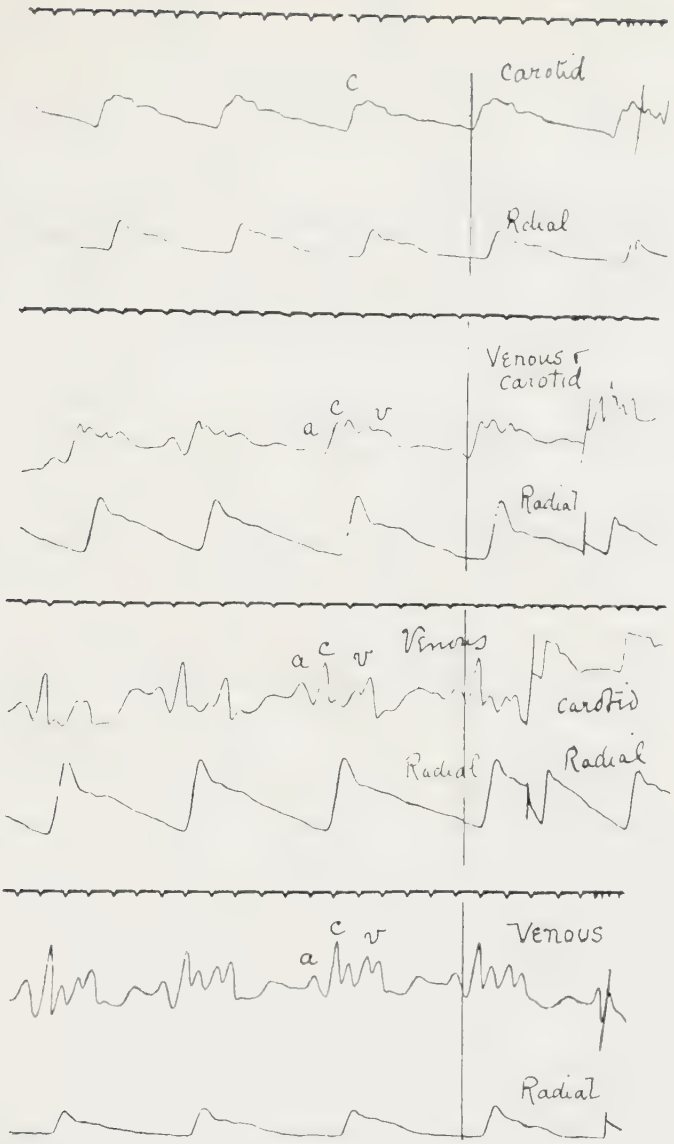


Fig. 86. This figure includes four polygraphic tracings taken from a single patient. The lower curve in each strip is from the radial artery. The upper curve in all tracings is from a single and fixed point in the neck. The differences in the neck curves are dependent upon the pressure at which the receiver was applied. In the uppermost curve the pressure was heavy; in the lowermost curve light; in the middle curves moderate. The figure shows transitions from the arterial to the venous type of curve as pressure upon the neck is relaxed and the receiver recedes from the artery. The first tracing exhibits a curve of purely arterial character; the second tracing demonstrates a small *a* wave directly to the left of the carotid upstroke; the third tracing shows an almost perfect, the fourth a fully developed, phlebogram. Vertical lines have been drawn at approximately corresponding points in the four tracings.

The figure illustrates a simple method of proving the precarotid time-relation of the wave *a*.

The interpretation of curves in which there are disturbances of sequence.

Since the identification of "*a*" waves is the aim of venous pulse work, no wave should be so lettered until all other sources of wave production at the given instant are excluded.

The interpretation of abnormal curves hangs largely upon our knowledge of experimental irregularities. Where the usual form of *v* is modified from point to point in a curve, and where an unusual and synchronous event is observed in the arteriogram, the source of *v*'s variation should be sought diligently. If the amplitude of a given *v* appears to be exaggerated, it may be suspected that an auricular systole has fallen during the ventricular systole, a view which is confirmed if the time-relations of the suspected *v* wave are not the customary ones (Fig. 91).

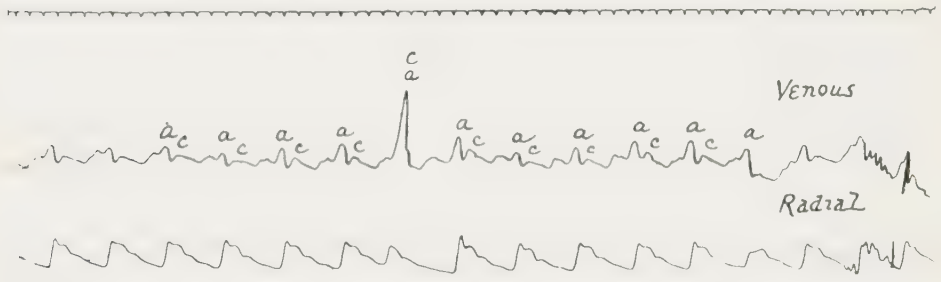


Fig. 87. Polygraphic curves from a patient, showing a premature contraction of the ventricle. The premature beat of the ventricle is seen in the radial curve. Simultaneously, a prominent summit ($\frac{c}{a}$) occurs in the venous tracing. This exaggerated wave has been produced by synchronous contraction of the auricle and ventricle, the auricular contraction falling in its place to complete the rhythmic series of auricular systoles while the ventricular contraction is premature.

Prominent summits.

When, from time to time, prominent summits occur in the jugular curve, and when such summits are notably larger than any of the waves accompanying cycles of the normal rhythm of the same case, it is probable, in the absence of variation in the amplitude of venous waves as an accompaniment of respiration, that at such points auricle and ventricle have contracted together, and that the auricle, instead of emptying itself into the ventricle, has created a sudden increase of venous volume (*261*, *501*, *624*).

Frequently it will be found that auricular systoles are expected at the points at which the exaggerated peaks fall and that abortive ventricular contractions are present at corresponding points in the radial or in apical curves (Fig. 87).

When an auricular beat, one of the rhythmic series, is expected at the instant at which the prominent summit appears, the meaning of the exaggeration is always clear. But similar waves sometimes spring up in the jugular curve and are simultaneous with abortive ventricular beats, even when an auricular systole is not expected. In such circumstances (Fig. 88) it may still be assumed that the auricle has contracted; for a relatively weak ventricular contraction, standing by itself, is not accompanied by an increased, but by a decreased wave *c* in the venous record. The simultaneous contraction of auricle and ventricle is due in such instances to prematurity of the contraction in both auricle and ventricle a disturbance termed a premature beat of *A-V* nodal origin or a *nodal extrasystole.*

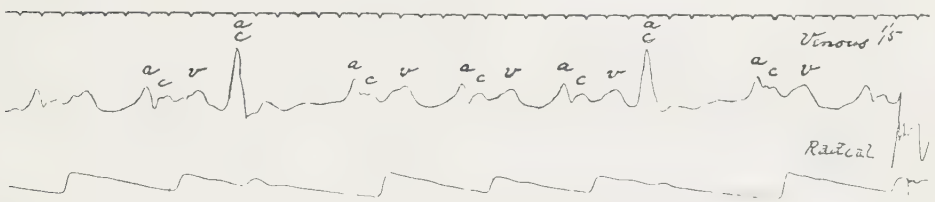


Fig. 88 A polygraphic curve from a patient who exhibited premature contractions of *A-V* nodal origin. Two such are shown, and are displayed in the radial curve by early and weak ventricular beats and in the venous curve by exaggerated summits ($\frac{a}{v}$) which occur earlier than expected *a* waves. Another curve from this patient is shown in Fig. 192, page 231.

Thus, exaggerated waves of the kind discussed are encountered when a premature ventricular systole synchronises with a rhythmic auricular systole, or with a premature auricular systole. A similar, though less conspicuous exaggeration happens within the limits of a rhythmic ventricular contraction when the next auricular systole falls so early that it coincides with the last phase of a ventricular systole. This may eventuate when the auricular systole is premature, the premature *a* falling with the *v* of the preceding ventricular contraction (Fig. 89). Collision between auricular

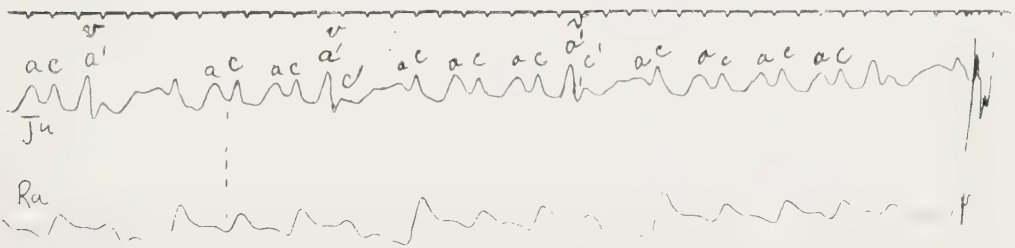


Fig. 89. A polygraphic curve from a patient showing three prominent waves $\frac{a}{v}$ resulting from premature contractions of the auricle; the exaggeration is rarely so great, in the event of *a* and *v* coinciding, as in the event of *a* and *c* being synchronous.

and ventricular contractions is also seen in many instances of rapid heart action, ventricular diastole being so curtailed that auricular systole is not confined to this portion of the ventricular cycle. In these cases the exaggeration occurs at each beat of the heart (Fig. 90). Widening of the *a-c*

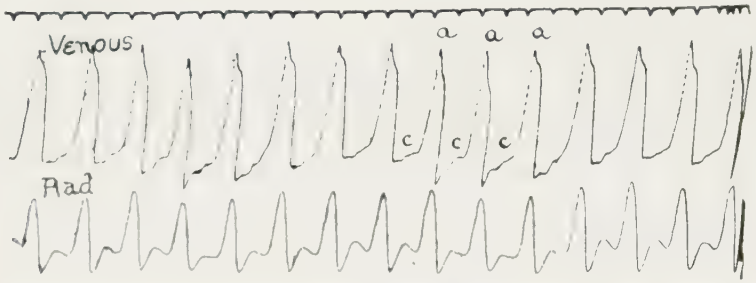


Fig. 90. Polygraphic tracing from a patient during a paroxysm of rapid heart action. The venous waves *a* are unusually prominent because they fall within the confines of the preceding ventricular systoles (ventricular systole in the jugular curve begins at *c*, and *a* comes almost immediately afterwards). The *a-c* interval in this instance is increased.

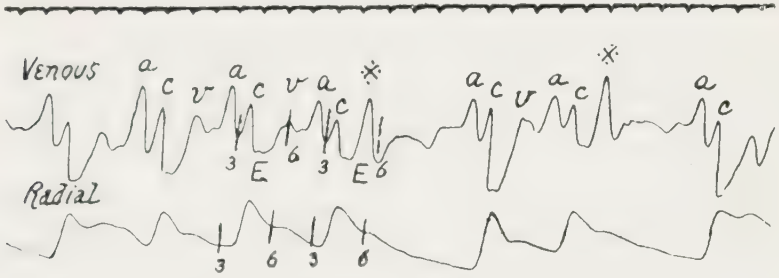


Fig. 91. A clinical polygraphic curve showing two premature contractions of auricular origin. The radial pulse is regular at first and is then disturbed by two unusually long pauses. Opposite each pause an exaggerated wave marked with an asterisk is seen in the venous curve. These two waves are of like nature. To take the former as an example; it might seem natural to assume this wave to be the *v* wave of the corresponding ventricular systole; but it is much larger than the last *v* wave and differs from it in form. These features should themselves draw attention to the wave in question; especially if, as in the present instance, it is repeated whenever the unusual pause is seen in the radial tracing. Its nature is determined to be auricular, when its relations to ventricular systole are examined. The latter begins in the jugular on line *3*, it ends at line *6*, lines which coincide with the beginning of *c* and the summit of *v*. But the line *6* for the particular cycle considered falls clear of the wave marked by an asterisk. The latter is therefore no *v* wave and must have been produced by an interference, such as contraction of the auricle. Further and conclusive evidence is to be found when the auricular contractions are spaced, for it is then ascertained that the dominant (auricular) rhythm has been disturbed.

interval is partly responsible for the coincidence of contraction in many of these instances, so that even with little acceleration of the heart's action an auricular contraction of one generation may fall with the ventricular contractions of the preceding generation, as Wardrop Griffith has aptly described

Fig. 92-94. Three curves from one patient and taken at one sitting. They illustrate changes in the jugular curve resulting from alterations of the *a-c* interval.

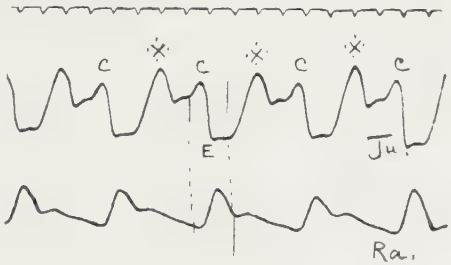


Fig. 92. In this curve the *c* waves in the jugular were readily determined in the usual fashion. The remaining waves are those marked by asterisks. What is their origin? Are they *v* or *a* waves? If the limits of ventricular systole are ascertained approximately, these waves are found to lie in ventricular diastole, rising to summits which lie nearly two-fifths of a second after the end of the ventricular systole. They are *a* waves, therefore, and the *a c* intervals are prolonged to nearly half a second's duration.

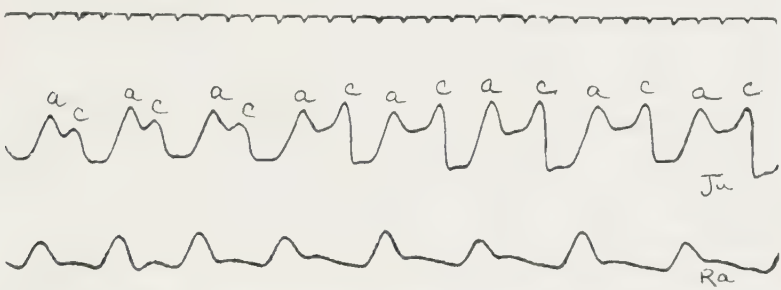


Fig. 93. From time to time a gradual widening of the *a-c* interval was witnessed, and this figure illustrates the gradual change of *a* from its usual presystolic position back to a position in mid-diastole. The heart's mechanism for the last few cycles of this figure is identical with that illustrated in Fig. 92.

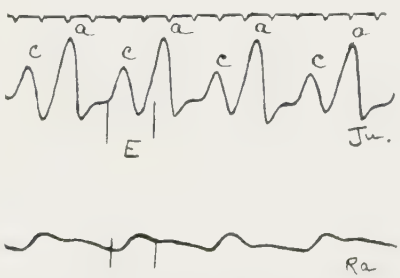


Fig. 94. The widening of the *a-c* interval sometimes proceeded further, the ventricular systole being so far delayed that it coincided partially with the following auricular contraction. This condition of the heart might be continued for many cycles. It is illustrated in this figure, which should be compared with Fig. 92. The tall wave *a* of Fig. 94 lies at its commencement within the period of ventricular systole (period *E*), its summit probably lies just beyond the opening of the auriculo-ventricular valves. Its exaggeration as compared with the corresponding wave of Fig. 92, is due to partial coincidence of auricular and ventricular systoles. The *a-c* interval in Fig. 94 has reached half a second.

the phenomenon. But coincidence is also witnessed when the heart is accelerated, though the *a-c* intervals are normal in length.

To illustrate the importance of exaggerated waves and the timing of these, I publish several special figures (Fig. 91-94), which are described in detail in the accompanying explanations.

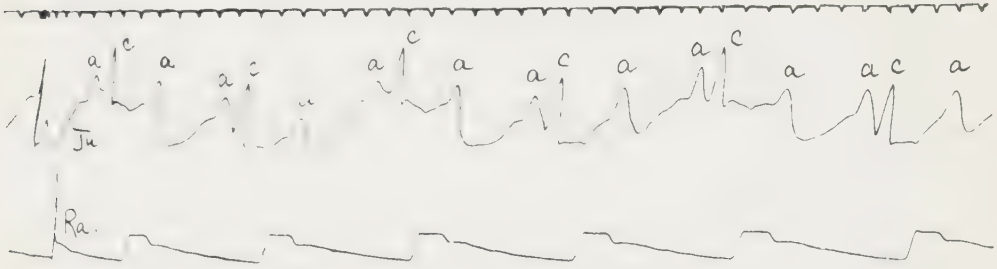


Fig. 95. Polygraphic curves from a clinical case. The *c* waves are each preceded by presystolic waves *a*, and there are certain additional waves of similar appearance, falling outside the limits of ventricular systoles, and placed equidistantly from preceding and succeeding *a* waves. They are also the result of auricular contractions. The auricle is contracting at exactly double the rate of the ventricle.

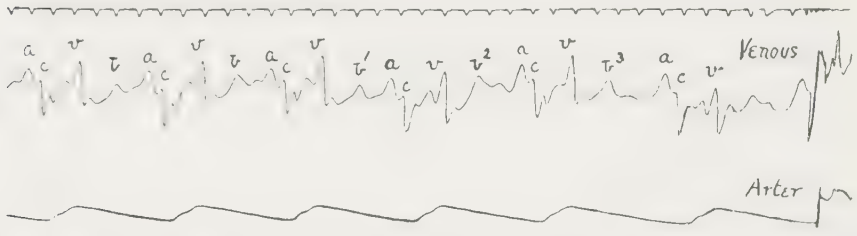


Fig. 96. Venous and arterial curves from a boy in whom the heart's action was slow and somewhat irregular. Each heart cycle is accompanied by *a*, *c*, *v* and *b* waves; the intervals between *v* and *b* are constant, between *b* and *a* they are inconstant.

Supernumerary waves.

If in a curve from a patient, where three clearly inscribed waves accompany each ventricular systole (waves which may be identified as *a*, *c* and *v*), a fourth wave occurs in the diastolic period of each cycle, it may be due to an auricular systole yielding no ventricular response. The interpretation is confirmed if the fourth wave is of similar form to the known *a* waves in the same curve, and if it is placed equidistantly between the preceding and succeeding *a* waves (Fig. 95). In these circumstances the pulse is generally slow. When the fourth wave is inconspicuous, and particularly when the remainder of the curve shows no evidence of impaired conduction, it may possess a different origin.

When the pulse is slow, a physiological wave, described by A. G. Gibson (*225*), is sometimes found in mid-diastole (see page 29). He termed it *b*, and is not difficult to recognise if different heart rates are studied in the same case ; sufficient change of heart rate may be obtained by slight exertion, or, in some instances, by the suspension of respiration. It is also easy to identify when the lengths of ventricular cycles vary naturally from time to time. In Fig. 96, there is in addition to the usual *a*, *c* and *v* waves of each cycle, a prominent wave in each diastole. But the pulse is not quite regular and the diastoles have a varying duration. If the positions of the supernumerary waves, relative to the preceding *c* and *v* waves and relative to the succeeding *a* waves are noted, it will be found that, when diastole is short, the extra wave (b^2) lies much closer to the next *a* wave, than when diastole is long (b^3). But the supernumerary waves lie at practically a constant distance from preceding *c* and *v* waves ; they belong, then, to the preceding systole of the ventricle.*

Changes in the frequency of the heart rate or the chance occurrence of longer pauses in the ventricular rhythm are also helpful in discovering impaired conduction where the heart beat is regular for long periods and auricular and ventricular systoles are fused ; for at the longer pause the two waves separate. When in addition to clearly established *c* and *v* waves there remains a series of further waves, falling at regular intervals but bearing no fixed relation to ventricular systoles, the regular and added waves are due to auricular systoles (Fig. 97). In such instances, it will be noticed that where an *a* wave falls during the limits of ventricular systole, its prominence is enhanced (Fig. 97).

Records of premature beats.

In the interpretation of curves in which single premature beats are transmitted to the radial tracing, the points in the venous curve at which the ventricular systoles commence are fixed first of all. In rare cases it may be necessary to calculate the actual transmission interval for the premature beat (Fig. 98). If the wave *c* corresponding to the premature ventricular systole is immediately preceded by another wave, this last wave is due to auricular systole (Fig. 99). The presence of a premature auricular contraction is confirmed if the dominant rhythm of the heart is disturbed.

But if no such wave is found and an exaggerated peak occurs at the instant of ventricular systole, then the auricle and ventricle have contracted together (Fig. 87 and 88, page 144).

Absence or apparent absence of "a" waves.

When the venous curve presents no distinct *a* waves, and when the chief summits of the curve fall without exception within the limits of ventricular

* The nature of *b* waves was discussed on page 29.

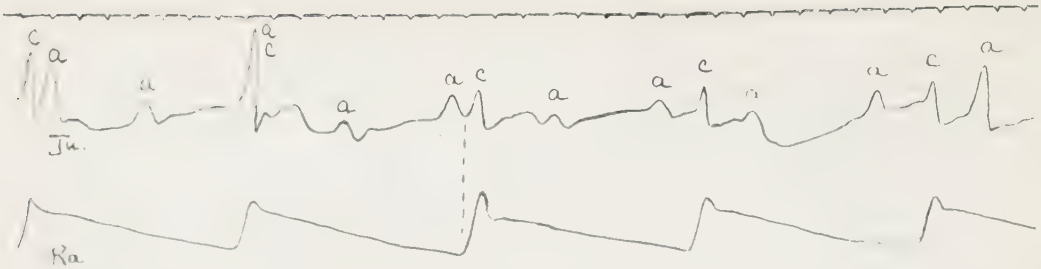


Fig. 97. Polygraphic curve from a case of Adams-Stokes' Syndrome. The *c* waves have been identified in the usual manner, and there remains a series of waves, scattered at approximately equal intervals in the curve, some of which are presystolic in time. They are all due to auricular contraction. Where, as happens in one instance, *a* and *c* fall together, a very exaggerated wave results. The auricular and ventricular rhythms are dissociated; the auricle is beating somewhat more than twice as rapidly as the ventricle.

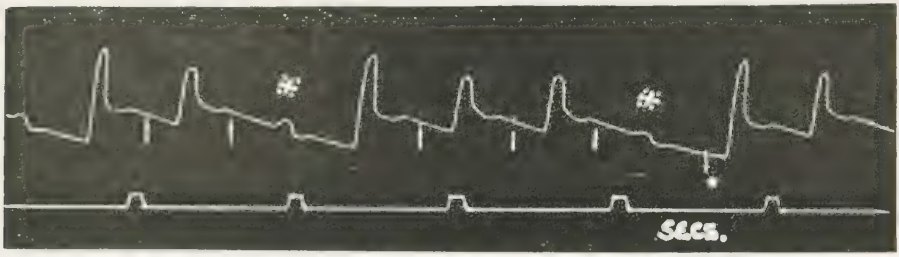


Fig. 98. A curve taken from a dog's carotid, by means of a Hürthle manometer. It shows two weak pulsations marked with asterisks. These resulted from premature contractions arising in the ventricle. Yet the carotid beats are not premature, but delayed. The increased delay may be calculated from the short ordinates which represent the actual times of onset of ventricular systoles, determined from a simultaneous curve taken direct from the ventricle. This fallacy in estimating the onset points of ventricular systoles from arterial curve is one which should be kept in mind (*256*), though it is but rarely responsible for an inaccurate interpretation.

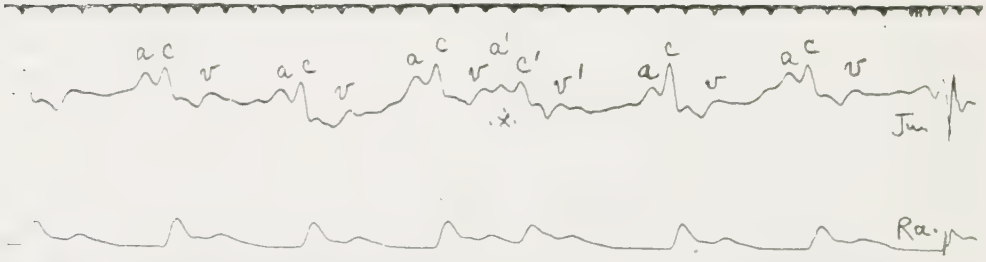


Fig. 99. Clinical polygraphic tracing, in the radial curve of which a single premature beat occurs. It is accompanied by waves *c'* and *v'*. Each of the rhythmic beats is associated with *a*, *c*, and *v* waves. An additional wave (*a'*) at the point marked with an asterisk is attributed to premature contraction of the auricle.

systole, the curve exemplifies the *ventricular form of venous pulse*. This form of venous pulse is encountered in many different circumstances. The arterial pulse accompanying it may be regular or irregular.

A. When the arterial pulse is *regular* the ventricular form of venous pulse is due to one or other of the following conditions :—

(*a*) engorgement of the auricle (Fig. 100). In such curves, *c* and *v* tend to fuse and form a plateau ; the movements of the distended auricle are incapable of causing a noteworthy impression upon the curve.

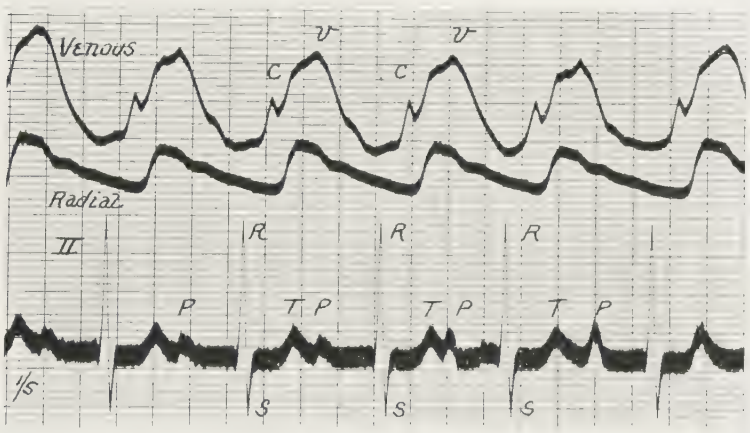


Fig. 100. Venous, radial and electrocardiographic curves from a case of cardiac failure with engorgement of the auricles. The venous curve is of the plateau variety and the *a* waves are not at all distinct. Yet the electrocardiogram shows that the auricle is contracting in early diastole. Time in fifths of a second.

(*b*) simultaneous contraction of auricle and ventricle, which may itself be produced in one of several ways (*4* and *446*).

1. Auricular contractions falling with ventricular contractions of the preceding generation (long *a-c* interval) (*4*, *762*).

2. Retrograde contraction of the chambers (*434*) (see Chapter XXI).

3. Simultaneous response of auricle and ventricle to impulses formed in the *A-V* node (see Chapter XV).

(*c*) slow and regular action of the ventricle, associated with fibrillation of the auricle (a condition fully described in Chapter XXV).

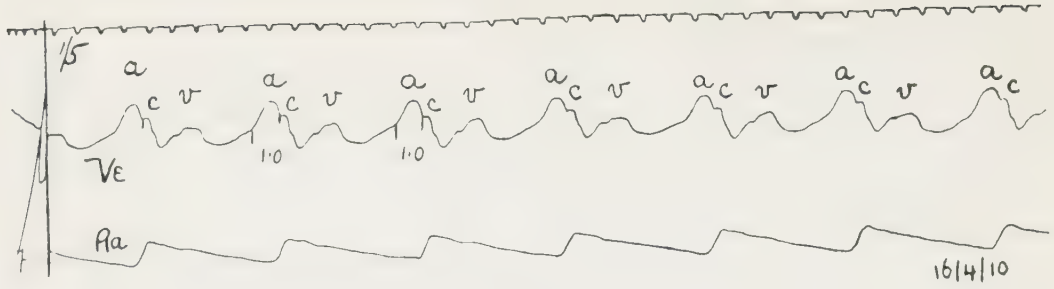


Fig. 101.

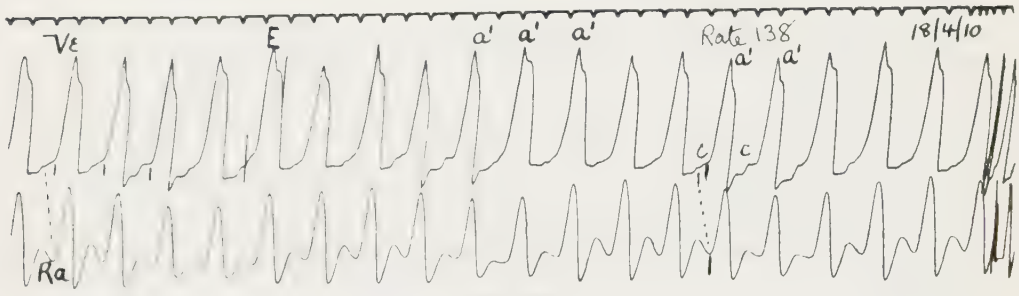


Fig. 102.

Fig. 101 and 102. (*Heart, 1910-11, II, 130, Fig. 1 and 2.*) Two polygraphic curves from a case of paroxysmal tachycardia. The first figure shows the normal curves of the slow periods. The second figure shows the ventricular form of venous pulse. The prominent waves fall during the period *E*, which marks the limits of ventricular systole. Resulting from simultaneous contraction of auricle and ventricle.

B. When the arterial pulse is *irregular*, the ventricular form of venous pulse is almost always due to fibrillation of the auricles (see Chapter XXIII).

Chapter XII.

The Analysis of Disordered Mechanism.

Electrocardiographic Curves.

The analysis of most disorders of cardiac mechanism may be accomplished by studying arterial or polygraphic curves, and the principles underlying these methods are summarised in the two preceding chapters; they are practical diagnostic methods. The final path to accurate knowledge is the electrocardiographic method. For purposes of investigation its precision is unrivalled. It presents numerous advantages over other methods. The curves are directly inscribed by the muscle of heart chambers whose activity it is desired to record. The auricular and ventricular systoles* are clearly depicted and their relations to each other are accurately expressed. The curves are uncomplicated by transmission intervals. One fibre yields the double record, and transference of measurement from one curve to another is obviated. The electrocardiogram displays all the events in auricle and ventricle. Except on rare occasions, it provides a full analysis of the disorders examined. When, as sometimes happens, certain auricular complexes are obscured, it may be necessary to add a simultaneous record from the veins. The electrocardiograph not only records the activation of auricles and ventricles; it speaks clearly of the direction of the excitation wave in these organs. It has been repeatedly laid down that the form of the galvanometric curve, corresponding to the activity of a given muscle mass, is controlled by the order in which the muscle elements become excited. This, the first grammatic rule of a new language, is to be borne constantly in mind.

If the left ventricle is excited at its apex, the corresponding axial electrocardiogram is in the main diphasic (Fig. 103) consisting first of a downward phase and finally of an upward phase. If the right ventricle is stimulated at its base, the curve is of a different type; the first phase is upwardly directed and the second phase is downwardly directed (Fig. 104).

It is true that the form of an electrocardiogram may be affected in a minor degree by change in the position of the heart relative to a given pair of electrodes; it is also true that hypertrophy by producing preponderance of one or other chamber may influence the amplitude and even the direction

*The term systole is used as a convenient expression, the actual record is, of course, not of contraction but of the electric change which very slightly precedes and accompanies systole.

Fig. 103.

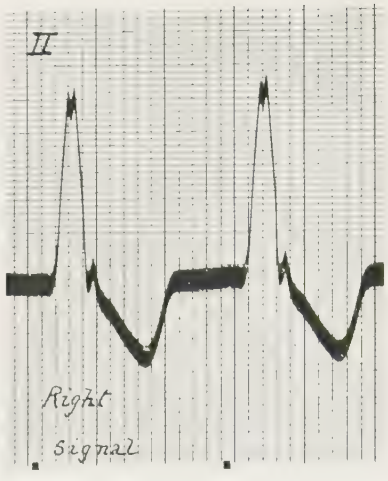


Fig. 104.

Fig. 103. Electrocardiogram from lead *II*. A dog's heart is responding to rhythmic stimuli (see signal marks at bottom of each curve) applied to the apex of the left ventricle. Time in fifths and twenty-fifths of a second.

Fig. 104. A similar curve from the same animal lead, showing the effects when the stimuli are applied to the conus arteriosus. These two figures illustrate the rule that the direction of spread regulates the shape of the electrocardiogram, which is consequently dependent upon the point at which the impulse originates.

of deflections. The curve is outlined from moment to moment by the direction of spread relative to the lead, and by the energy of the combined electrical discharges; but as the anatomical relations of the heart to a customary lead do not often vary very materially even in pathological conditions, and as the changes in the curves form this cause and from an abnormal balance of the right and left musculature are rarely profound, the direction in which the excitation wave spreads and is distributed constitutes the chief factor.

Auricular and ventricular contractions of physiological types.

Ventricular contractions of supraventricular origin.—When an impulse descends to the ventricle through the *A-V* bundle and is distributed through the uninjured arborisation of Purkinje, the excitation wave spreads normally throughout the ventricle. The corresponding electrocardiogram invariably consists of a group of initial deflections (*Q*, *R*, *S* group) which represents this normal spread, followed after an interval by a more or less prominent *T* deflection. It is a matter of indifference whether the impulse originates in the normal pacemaker, in the substance of either auricle, in the

A-V node or in the bundle down to the point of its subdivision; the spread in the ventricle is precisely the same, the resultant ventricular complex is the same, in each instance. Whenever the form of the natural electrocardiogram is known for a given patient, any systole of the heart which yields an electrogram of this type is promoted by what is termed a *supraventricular impulse*. That is to say, its impulse arises above the point of division of the *A-V* bundle and is consequently distributed to the ventricle in a fixed and orderly manner. This rule is absolute, whether the beat in question belongs to a rhythmic series or is the cause of an irregularity. Even when the normal electrocardiogram is not available for comparison, if the ventricular complexes—individual ones or all—conform to the general type of natural complexes, their supraventricular origin may be assumed.

The converse conclusion, that a ventricular systole of supraventricular origin will yield a natural electric curve, usually holds good for pathological as for normal conditions; but as will presently be seen there are important exceptions.

Beats arising in the vicinity of the pacemaker.

If a ventricular complex of supraventricular type is preceded by an auricular complex of normal outline a rounded or peaked summit—the spread of the excitation wave through the heart as a whole may be assumed to be natural and the origin of the beat may be stated to be from the vicinity of the pacemaker. Clearly this conclusion is especially serviceable when applied to isolated systoles the origin of which is to be ascertained; and it is absolute if the form of the electrocardiogram for such beats is in all detail a replica of beats known to be physiological in the same subject.

Anomalous beats.

When those minor changes in the complexes associated with malposition of the heart or hypertrophy of its chambers are excluded, departures from the natural type are the result of a single cause. This cause is abnormal spread of the excitation wave. As a natural electrocardiogram portrays a natural spread, so, with the exceptions named, an anomalous electrocardiogram portrays an unusual distribution of the excitation wave. This unusual distribution may be confined to auricle or to ventricle.

When it occurs in the auricle it is due to an unusual starting point of the heart beat. If systoles are propagated serially from the pacemaker, and this series is disturbed by a systole arising in some other portion of the auricular musculature, all the ventricular complexes of the curve will be alike, but the auricular complex corresponding to the disturbance will be of unusual form (Fig. 105). Arising in a new focus the excitation wave is propagated along abnormal auricular channels and a change in the corresponding portion of the electric curve results; but as the focus is

supraventricular the spread to the ventricle takes place through normal channels and the succeeding ventricular complex therefore suffers no distortion.

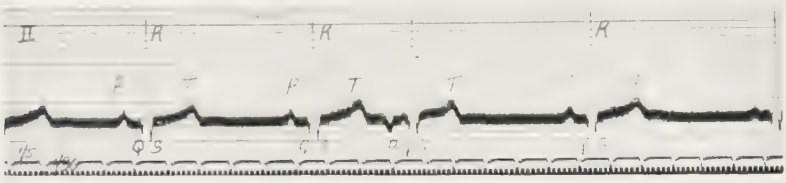


Fig. 105. A clinical electrocardiogram, showing a normal heart rhythm, is disturbed by a premature beat (the fourth). This fourth systole presents a ventricular complex of normal outline; it has arisen therefore in a supraventricular focus. It is preceded by an auricular complex which is inverted, showing that the excitation wave has taken an abnormal course in the auricle and that the corresponding impulse arose in an abnormal situation in the auricle. Time in thirtieths and fifths of a second.

If the systole disturbing the normal series is propagated from the ventricle, the corresponding ventricular complex is altered (Fig. 106), because, when the excitation wave starts at any point below the bifurcation of the bundle, the spread of the excitation wave in the ventricle is abnormal. While this is the cause of most anomalies in the ventricular complexes, the same explanation does not always hold good. For as the shape of the electric

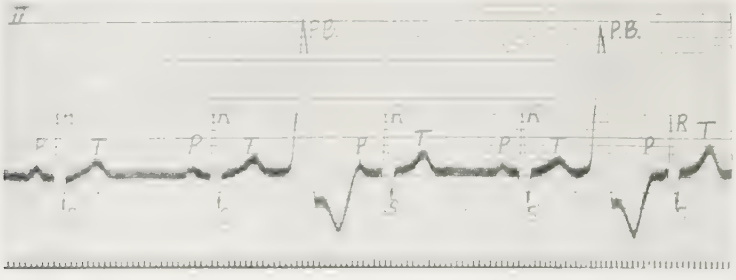


Fig. 106. A curve showing two anomalous beats interpolated between natural heart contractions in a patient. These beats arose at some focus in the ventricle, and the corresponding ventricular complexes are abnormal, since the excitation wave has pursued an abnormal course in the ventricular muscle. Time in thirtieths of a second.

curve is governed by the direction in which the excitation wave travels, this direction may be modified, not only by an alteration of the point from which the contraction originates, but by changes in those special channels of conduction which exist in the ventricle (Fig. 107). An impulse entering the ventricular segment of the cardiac tube through the auriculo-ventricular

bundle passes into the two bundle divisions; but conduction may be defective in one of these, or it may be defective in more distal branches of the

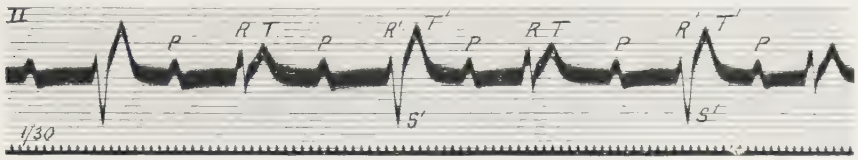


Fig. 107. An electrocardiogram taken from a cat during asphyxia. The ventricle responds to each regular auricular impulse (P); all the ventricular beats are therefore of supra-ventricular origin; but the first, third and fifth responses of the curve are unnatural. These complexes show deepening of S and lifting of T, changes which are due to aberration. Time in thirtieths of a second.

arborisation. The course of spread in these circumstances is *aberrant* (see Chapter IX) and according to the degree of aberration the electric curve is correspondingly modified. So far as we know "aberration" is peculiar to the ventricle; the auricle is exempt because in its structure it possesses no special conducting paths.

Identifying different types of anomalous contraction.

If an electrocardiographic curve exhibits beats of physiological type, and beats of anomalous type occur as occasional interruptions, the contrast between one type and the other is generally conspicuous and the anomalous beats are at once recognised. But though the dissimilarity is usually manifest, the custom of comparing the anomalous beat with those of physiological type in the same case, cannot be too strongly urged. For what is a somewhat anomalous type in one case may be physiological in another; the departure from the physiological type is at times inconspicuous; this is the case where there is but slight alteration in the spread of the excitation wave.

The cause of a given anomaly is usually known as soon as the curve is inspected, for the common anomalies are characteristic and speedily become memorised. Nevertheless, each curve should be treated on its own merits.

When we deal with a premature beat, the ventricular complex may be anomalous. In that case, and if it is not preceded at a natural interval by an auricular systole, the ventricular origin of the beat is known. If the ventricular complex is natural in outline and is preceded by an anomalous auricular complex, we recognise that a new focus in the auricular muscle has originated the disturbance. But if the ventricular complex is abnormal, and at the same time it is preceded by an auricular complex of unusual type, then not only is there a disturbance in the site of auricular impulse formation, but the paths of conduction through the ventricle are also defective (see Fig. 112*a*).

Aberrant contractions—which as previously stated are confined to the ventricle—may be identified if they conform to a recognised type; or if, departing in form from physiological beats in the same subject, they are preceded by auricular systoles, for these betray their supraventricular origin. The chief forms of aberrant contraction are described in a previous chapter; other forms will be described at a later stage.

The algebraic summation of complexes.

In many examples of disordered mechanism it happens that ventricular and auricular systoles fall synchronously, and in discussing venous curves, we saw that simultaneous contraction of the upper and lower chambers produces a special form of phlebogram, dependent upon hindered discharge of the auricular contents.

Individually the electric complex of the auricle and of the ventricle are not appreciably changed by such coincidence of the contractions, and the curve which results is consequently a simple composite of auricular and ventricular complexes. The auricular systole may fall at any point upon the ventricular systole, and its representative (P) will be found, superimposed upon R, S or T. The summation of effects is exact algebraically.

When complete dissociation of the auricular and ventricular rhythms is present, the deflections P are of normal form and the deflections R, S and T show a close resemblance to the normal type. Auricular complexes, representing beats arising in the vicinity of the pacemaker are superimposed upon ventricular complexes of supraventricular type (Fig. 108).

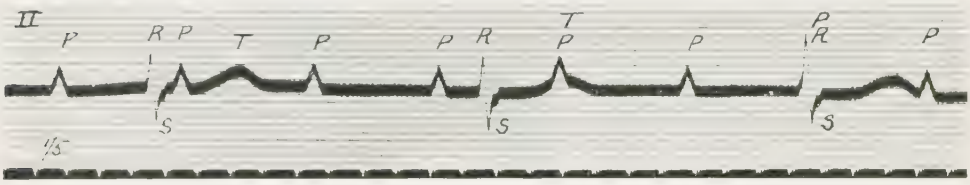


Fig. 108. A curve from a patient who displayed complete heart-block, or dissociation of the auricular and ventricular rhythms. The auricles and the ventricles are beating regularly, but at the independent rates of 78 and 29 per minute, respectively. The exact superimposition of auricular and ventricular summits should be remarked. Time in fifths of a second.

If the individual cycles from such curves as those of Fig. 108 are excised, they may be re-arranged above each other, not in the order in their natural sequence, but in an order which renders the superimposition more manifest. Such a re-arrangement is seen in Fig. 109. Traced from above downwards, the first auricular summits pass gradually into,

through and beyond the opening phases of ventricular systole; the second row of auricular summits continues the tale, and shows the passage over and clear of the broad summit *T*. The summation is always exact; the height of *R* in the fifth curve of this figure is especially noteworthy: the amplitude is that of the natural *R* and *P* combined.

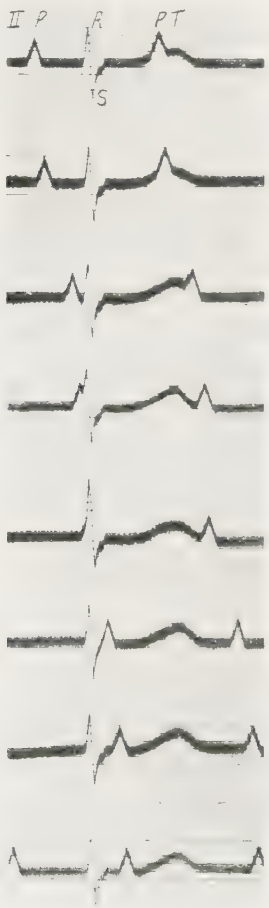


Fig. 109. A figure constructed from the electrocardiograms of the patient whose curve is shown in Fig. 108. Single ventricular complexes have been rearranged above each other, not in the order of their natural sequence, but so as to display the varying relation to them of the auricular complexes.

When a premature ventricular contraction coincides with a rhythmic auricular systole, a *P* of normal type is superimposed upon an anomalous ventricular complex (Fig. 110 and 111). It is not always easy to identify such auricular beats, for the exact and uncomplicated form of the anomalous

ventricular complex may not be known. But where the prematurity of the ventricular contraction is variable, even if only slightly variable, as in Fig. 110, the auricular portion of the curve can often be identified by noting the slight differences in outline between the two anomalous ventricular complexes. The buried auricular summit, when found, is seen to occupy a midway position between preceding and succeeding auricular contractions.

The buried auricular summits can also be identified when two premature beats occur in succession, for here too the anomalous ventricular complexes vary, the auricular summit falling with one only, and falling equidistantly between neighbouring auricular deflections (Fig. 111).

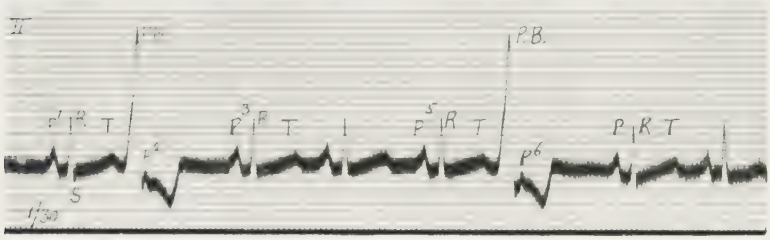


Fig. 110. A clinical electrocardiogram showing two premature contractions of ventricular origin. The auricular contractions are regularly dispersed throughout the curve; but two of them (P^2 and P^5) are buried in the anomalous ventricular complexes. That these summits are not part of the anomalous complexes is proved by the slightly different time-relations in the two instances. Time in thirtieths of a second.

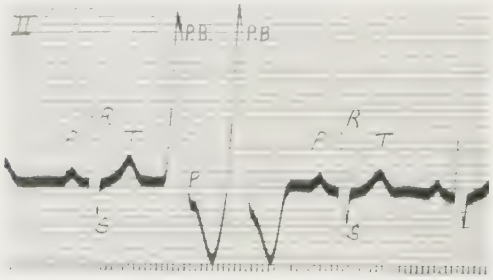


Fig. 111. Two premature contractions arising in the right ventricle of a patient. They occur together and replace a single normal ventricular contraction. The rhythmic auricular contraction falls with the first premature beat. Time in thirtieths of a second.

When a premature and anomalous auricular contraction coincides with a rhythmic ventricular systole, an anomalous P is superimposed upon a ventricular complex of normal outline (Fig. 112*a* and *b*).

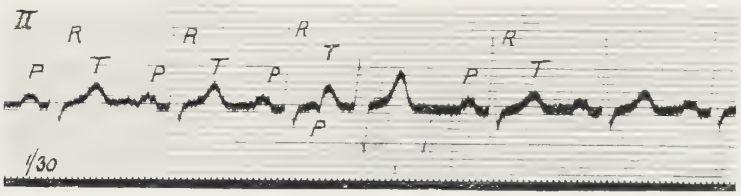


Fig. 112*a*. A premature contraction of auricular origin in a patient. The first three cycles are normal, but *T* in the last of these is notched at its beginning by the premature *P* which is of inverted form (compare the 2nd and 3rd *T* summits of the figure). This premature *P* is followed by a ventricular complex which differs from the normal on account of "aberration" in the ventricle. All the beats are of supraventricular origin. Time in thirtieths of a second.

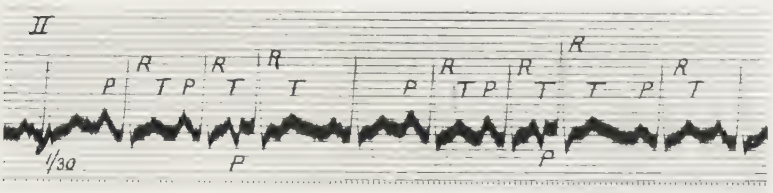


Fig. 112*b*. Two premature contractions arising in the auricle are shown in this patient's record. The auricular complex *P* is inverted and notches the preceding *T* in each instance. The ventricular complex of the second premature beat shows slight aberration. Time in thirtieths of a second.

The original observations upon which the principles of analysis discussed in this chapter are based, are described for the most part in the following original publications (*111*, *390-392*, *428*, *433*, *445*, *447*, *463*).

In the preceding chapters I have dealt with the principle rules guiding the analysis of disordered mechanism and have spoken of the more important phenomena observed in studying arterial, venous and electrocardiographic curves respectively. The observations outlined in these chapters will serve as a basis in proceeding to examine the chief disorders of the heart beat as they are met with in clinical work. Thus, while the first section of this book is devoted to anatomy, physiology and method, the second section will be devoted to the disorders themselves and to correlating them with experimental findings. It will be my endeavour to present the experimental and clinical facts side by side, as far as possible, and to identify the naturally occurring disorder with that produced by deliberate interference with the heart in the lower animals. The second section will also contain the evidence upon which many of the conclusions of the first section, and in particular the general rules of interpretation, are founded.

CHAPTER XIII.

EXPERIMENTAL HEART-BLOCK.

In Chapter VI, we saw that the sequential contraction of auricle and ventricle depends upon the integrity of the auriculo-ventricular bundle, and that experimental lesions of this neuro-muscular tract produce heart-block. We have further concluded that the impulse is conveyed to the ventricle from this bundle through its branches and their arborisations. It is permissible to make the more sweeping statement that the proper sequence of contraction hangs upon the functional integrity of the junctional tissues as a whole. Unless there is an intact pathway, traversing the node, the bundle and one or more of its branches, the responses of the ventricle are unpunctual or fail.

When a clamp is applied to the bundle and compression is gradually increased, heart-block displays itself, as Erlanger (*143*) showed, in successive stages; these stages comprise:

(*a*) An increase in the interval between the onset of auricular and ventricular systoles; an increase of the *As-Vs* interval as it is termed, which in the normal dog approaches 0·1 sec..

(*b*) An occasional failure of the usual ventricular response to the regular auricular impulses.

(*c*) A failure of ventricular response to each tenth, ninth, eighth, seventh, sixth, fifth, fourth or third auricular contraction.

(*d*) A failure of the ventricle in its response to alternate auricular beats; the establishment of 2 : 1 heart-block in which the auricle beats precisely twice as rapidly as the ventricle (Fig. 113).

(*e*) The response of the ventricle to each third or fourth beat of the auricle; so-called 3 : 1 or 4 : 1 heart-block, of which the latter is by far the commoner in experiment.

The foregoing grades of block are spoken of as partial.

(*f*) The onset of complete heart-block (or dissociation) between auricular and ventricular rhythm, *i.e.*, entire failure of conduction (Fig. 114). Soon this is associated with a new event, namely, the wakening of a rhythm inherent in the ventricle.

Examples of heart-block produced by compressing the *A-V* bundle in dogs are shown in Fig. 113 and 114.

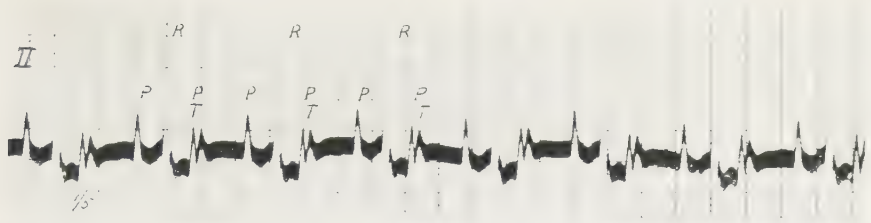


Fig. 113. ($\times \frac{5}{6}$.) The figure exhibits 2 : 1 heart-block, the result of lightly clamping the bundle in a dog; the auricle is beating twice as rapidly as the ventricle. Alternate auricular systoles, those to which the ventricle fails to respond, fall within the confines of ventricular systole; the corresponding P summits appear immediately prior to the T summits. Time in fifths of a second.

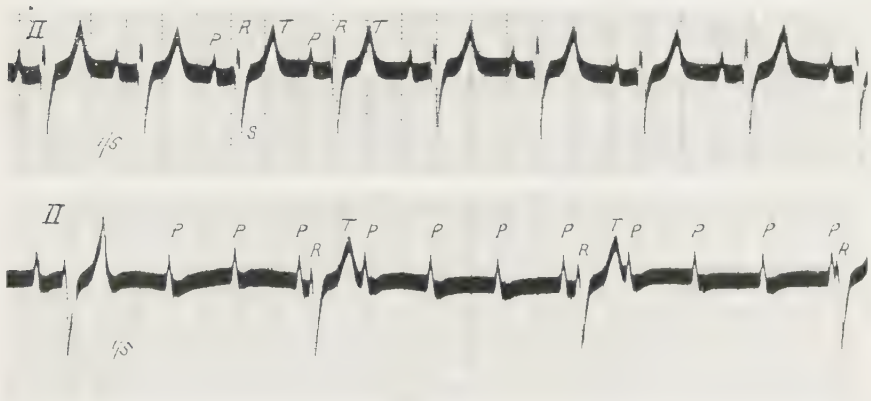


Fig. 114. ($\times \frac{5}{6}$.) Two curves from another animal; the first taken immediately after the clamp was applied; the second after firm compression. The top curve exhibits a slight prolongation of the P-R interval. The bottom curve shows complete dissociation; in this figure auricle and ventricle beat regularly but at independent rates and the relation of the ventricular cycles to adjacent or simultaneous auricular cycles is variable. These two curves are also published to illustrate the similarity of the ventricular complexes, before and after the dissociation. The right bundle division was also injured in this instance, whence the anomalous character of the ventricular electrocardiogram. Time in fifths of a second.

Methods of producing heart-block in animals.

Heart-block between the auricles and ventricles may be produced in one of three chief ways.

1. *By direct interference with the conducting tracts, namely, the auriculo-ventricular node, the bundle or both divisions of the latter*:—The experiments in which this tract has been injured by pressure or section have been described in detail. Heart-block may be produced more delicately by cooling this region of the heart (*41*, *797*).

2. *By stimulation of the vagus.*—That vagal impulses are capable of producing heart-block has been recognised since Gaskell's observations (*214*). In studying conductivity changes in the frog's heart, many workers have adopted almost exclusively this method of producing heart-block. The vagal tone may be increased reflexly by stimulating the gastro-intestinal tract (*127*). Chauveau (*47*), one of the earliest observers to record heart-block in the mammal, produced it in the dog by vagus stimulation.

Rothberger and Winterberg (*665*), working upon the dog's heart, suggested that the cardiac nerves are distributed in a special manner, it being supposed that the right vagus is distributed mainly to the *S-A* node, the left vagus to the *A-V* node. These writers state that weak stimulation of the right nerve usually produces slowing of the whole heart, while similar stimulation of the left nerve has a less conspicuous retarding action, but produces defects in *A-V* conduction. These effects of vagal stimulation have been amply confirmed (*57*, *60*, *466*). That the *S-A* rhythm is most readily inhibited by stimulation of the right vagus is undoubted; the difference between the two nerves is profound in this respect. But that *A-V* conduction is affected chiefly by stimulation of the left nerve is more open to question. The *apparent* difference (Fig. 115 and 116) is a profound one, but it is chiefly conditioned by the associated rates of auricular contraction. If a constant auricular rate is deliberately maintained in these experiments, the difference in the effects of the two nerves is less conspicuous; the left nerve appears, however, to be slightly, though not uniformly, preponderant in these circumstances (*74*).

The effects of vagal stimulation are not confined to the main bundle, an influence is exerted also upon the divisions of the bundle (*60*, *100*, *123*), for not uncommonly when partial heart-block is produced by vagal stimulation, responses of the ventricle of aberrant type are to be seen* (Fig. 117). There is, however, no definite relation between the side on which the nerve is stimulated and the division of the bundle which is affected.

3. *By introducing toxic bodies into the blood-stream.*—A very large number of toxic substances will produce varying grades of heart-block when they are injected into the blood-stream of animals. Amongst those which may be named are digitalis (*85*, *716*), strophanthine (*672*), aconitine (*87*), muscarine (*663*), physostigmine (*663*), nicotine (*594*), glyoxyllic acid, morphia (*123*), adrenalin (*360*), and potassium salts (*546*). Certain of these substances, for example morphia and adrenalin, are known to act through the vagus centrally, for the disturbances are abolished by section of the vagi; others such as the salts of potassium appear to act directly upon the musculature of the heart.

* These observations are originally experimental; Wilson has recently recorded instances in which aberrant curves were seen to follow vagal stimulation in the human subject (*783*).

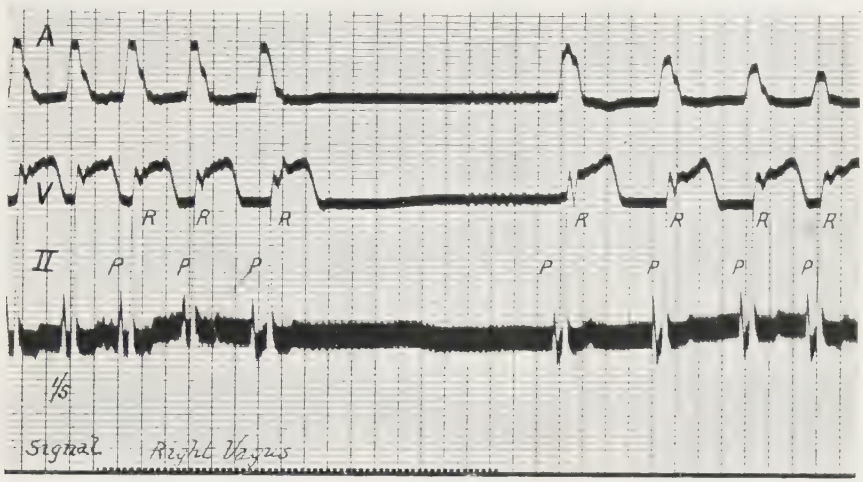


Fig. 115. ($\times \frac{10}{1}$.) Myocardiograms from auricle (A) and ventricle (V) and electrocardiogram, showing the customary effect of stimulating the right vagus in the dog. The P-R interval widens a little at first and then the whole heart ceases to beat. The signal of stimulation is seen below. Time in fifths of a second.

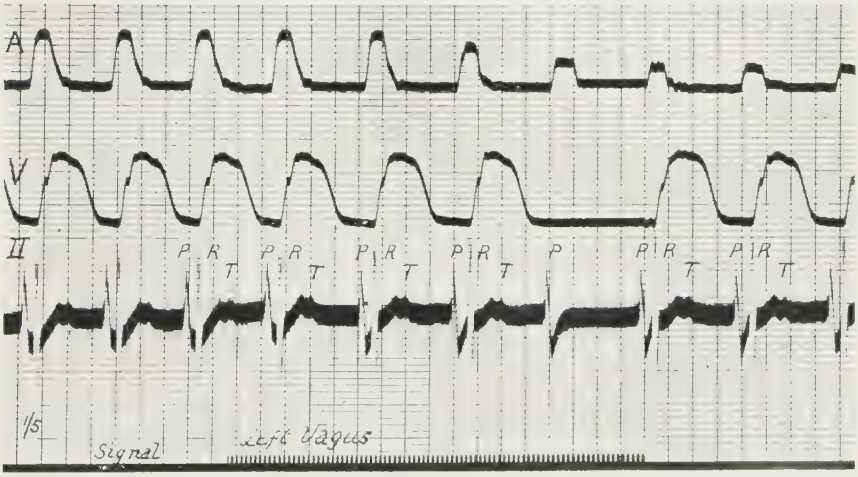


Fig. 116. ($\times \frac{10}{1}$.) Similar curves from another animal showing the customary effect of left vagal stimulation. The P-R intervals widen out, and the auricular rate being only a little diminished, the ventricle fails to respond. In this instance, only a single ventricular beat is missed. Time in fifths of a second.

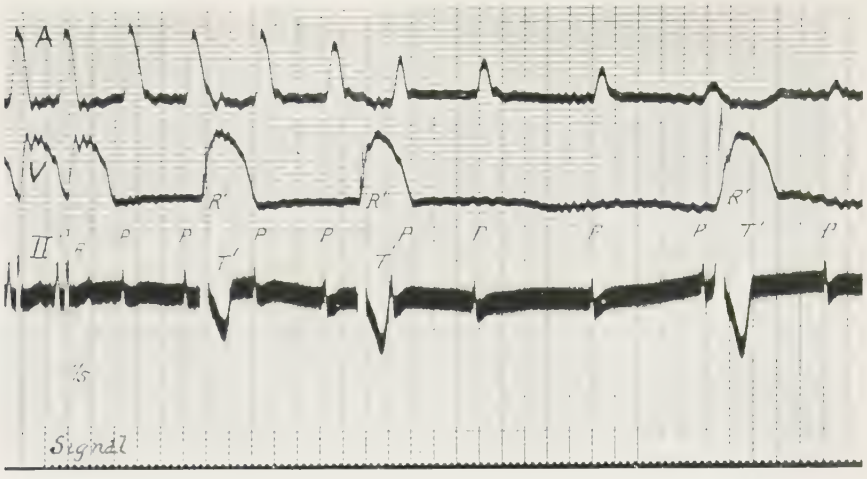


Fig. 117. ($\times \frac{9}{16}$.) Similar curves, showing an unusual effect of stimulating the right vagus in the dog. *A-V* heart-block results and the character of the complexes of ventricular responses alters in a manner indicating hindered conduction in the left division of the bundle. Time in fifths of a second.

Heart-block may be very readily induced in cats, less readily in dogs, by asphyxiating them (*481*, *699*). Such heart-block is independent of the vagus, for it occurs equally after full saturation with atropine or after vagal section (*481*); it is also independent of blood-pressure changes, of dilatation of the heart and of excessive accumulation of carbon dioxide in the blood (*545*); it probably results from lack of oxygen and is perhaps due to an excess of acid products in the blood. The heart-block may be of any grade, partial or complete (Fig. 118-121) but, when ventilation is restored, recovery is rapid and perfect, providing that the experiment is not carried too far.

The administration of fatal doses of diphtheria toxin is followed by *A-V* heart-block; the same failure of conduction is witnessed in death from anaphylaxis (*12*, *651*).

*Records of experimental heart-block.**

Records of the heart beat, subsequent to damage of the *A-V* bundle, or subsequent to other interferences productive of heart-block, may be taken in many ways; but we may confine ourselves at the present time to a brief description of the electric curves, for they are particularly legible. The curves obtained during asphyxia are used for illustrative purposes.

* While the experimental records of mammalian heart-block are described before the clinical for purposes of convenience, it is interesting to note that the majority of the observations were first made upon patients.

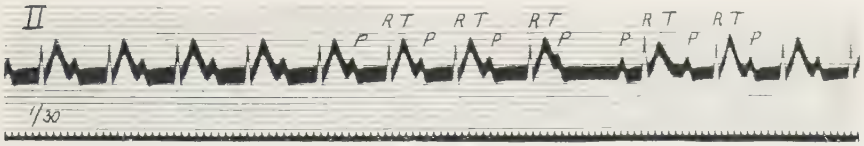


Fig. 118. ($\times \frac{4}{5}$.) An electrocardiogram from a cat, after one and a half minutes of asphyxia. In the first portion of the curve the *P-R* interval widens. This widening increases at the seventh cycle and the next *P* summit falls with *T* of the ventricle. A single response is missed, and the *P-R* interval then shortens up, gradually to widen once more as sequential contraction is resumed.

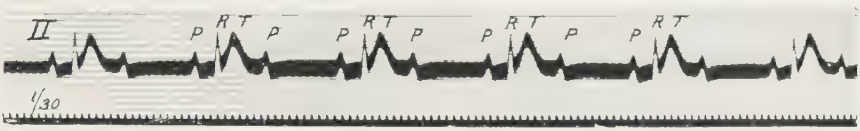


Fig. 119. ($\times \frac{4}{5}$.) A later stage of the same period of asphyxia; 2 : 1 block is established.

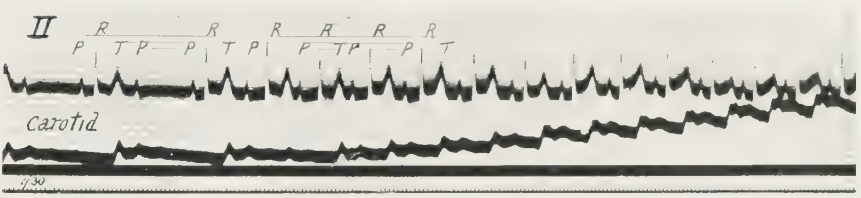


Fig. 120. ($\times \frac{4}{5}$.) After profound asphyxia in which dissociation developed, ventilation was re-established. The heart recovered completely. The curve shows the recovery from the stage of 2 : 1 heart-block to that in which there is at first some prolongation of the conduction interval. The simultaneous curve is a Hürthle manometer tracing of carotid pressure.

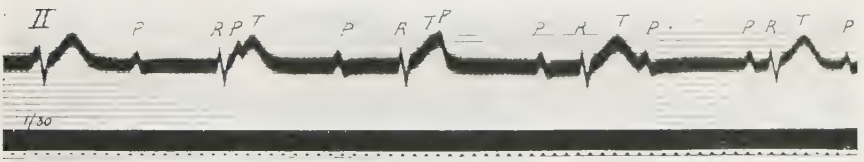


Fig. 121. ($\times \frac{4}{5}$.) A curve of complete heart-block, in which the rhythms of auricle and ventricle are each regular but independent. Resulting from profound asphyxia. Time in all this series of curves in thirtieths of seconds.

If a cat is asphyxiated for periods varying from 1-7 minutes, a regular succession of events is observed in the heart. Within 1-3 minutes of the onset of asphyxia the *P-R* interval (representative of the *As-Vs* interval) shows a notable prolongation. This phase may last for a shorter or longer time, and is succeeded by a condition in which single responses of the ventricle to auricle are missed (Fig. 118). At this stage the *P-R* interval shows changes of interest and importance. The *P-R* interval, which is primarily

increased, shows a gradual and further increase up to the point where the response is missed (Fig. 118); the delay in the ventricular response may be so great that a given ventricular contraction coincides with the following auricular contraction (the *P-R* interval in experiment may lengthen to 0·3 sec. at least). Subsequent to a failure of transmission and following the resultant ventricular pause, the interval decreases abruptly and the whole process is repeated. The alteration of the *P-R* interval changes the time relations of the ventricular beats. The dropping of a single ventricular beat is not accompanied by a pause equal to two heart cycles, but to a pause of shorter duration. Thus the grade of the irregularity in the ventricle is diminished. Minor changes in position occur in the remainder of the ventricular cycles; as the *P-R* interval increases, the rate of increase in *P-R* interval diminishes and the ventricular rate consequently quickens slightly.

The phase of prolonged *P-R* interval with dropped beats is succeeded by one of 2 : 1 heart-block, in which only alternate systoles of the auricle awaken ventricular responses (Fig. 119). Finally, complete dissociation develops, in which independent rhythms are found in auricle and ventricle, each regular, but bearing no simple mathematical ratio to each other (Fig. 121). In the electric curves the ventricular systoles (*R* and *T*) are represented at regular intervals in the curve, so also are the auricular systoles (*P*), but they fall in haphazard relation to the ventricular contractions. Where auricular and ventricular contractions coincide, accurate summation of the electric effects is noted.

The susceptible region.

When partial heart-block is produced by compression of the *A-V* bundle, the shape of the ventricular complexes does not change. This is no more than is to be expected, since the auricular impulses still flow along the accustomed channels and the distribution of the excitation wave to the ventricle is undisturbed. The curves are usually similar when partial heart-block results from vagal stimulation or from poisoning. It is almost inconceivable that the spread to the ventricle would remain unchanged as it does unless the vagus or the poison exerted its chief effect at a level of the conducting system where it is concentrated to form a single strand. For if the effect were produced more peripherally, and were not identical in its degree throughout the main stems of the conducting system, the spread to one part of the ventricle would be relatively quicker than to another, and a noticeable alteration in the form of the resultant curve would be seen. This argument suggests that the influence, nervous or toxic as the case may be, is a local one.

To take the vagus as an example, heart-block due to its over-action might conceivably be brought about by the influence of this nerve upon the conducting tract, or it might be supposed that its action is exerted upon the

ventricle, rendering this organ irresponsive. In this instance we are in a position to define its mode of action.

That the whole conducting tract is under some measure of vagal control seems clear, for under stimulation of one or other nerve, defects in conduction in the bundle divisions may be witnessed from time to time (Fig. 117, page 166); the complexes of the responding ventricle are anomalous, and the anomaly is recognised to result from aberration. But that the vagal influence does not produce *A-V* block by acting upon the bundle branches is suggested by the comparative rarity of branch conduction defects. Its influence is concentrated at a higher level of the system, and the complexes of the ventricle in the electrocardiograms remain unaltered in form. It has recently been shown by experiments specially devised for the purpose that the most susceptible point lies in the region of the junction between *A-V* node and auricular tissue (*466*). If heart-block is induced by stimulating the vagus, while the auricle and ventricle are responding simultaneously to impulses arising, not in the *S-A* node but in the *A-V* node, conduction to the auricle suffers in greater degree than does conduction to the ventricle. Likewise, in the heart-block of asphyxia, it has been demonstrated (*482*) that the same region suffers in highest degree and that, when conduction

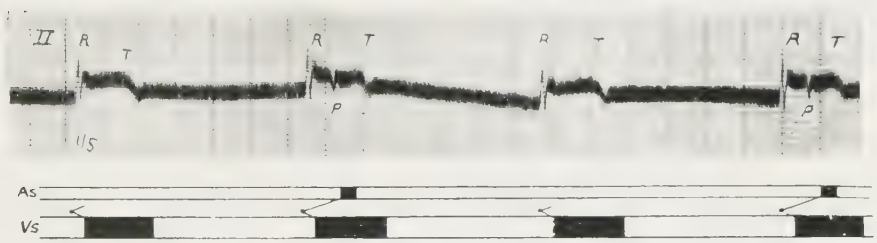


Fig. 122. (*Heart, 1913-14, V, 289, Fig. 11.*) (× $\frac{2}{3}$.) An electrocardiogram showing reversed heart-block. A cat was asphyxiated until the several grades of heart-block, shown in Fig. 118-121 were seen. After recovery it was again asphyxiated and at the same time cold was applied to the sulcus terminalis. The last procedure depresses impulse formation in the *S-A* node, and the *A-V* node then usurps its function (see Chapter XV). While this *A-V* rhythm is in progress, each impulse makes its way upwards to the auricle and downwards to the ventricle. It is the passage of the impulse upwards to the auricle which is affected by asphyxia, the response of the auricle at first lags by comparison with that of the ventricle. Eventually auricular response fails, while ventricular response continues. The present figure shows this failure of the auricular response at alternate beats. The two auricular complexes *P* which are seen in the figure are inverted (because the passage of the excitation wave through the auricle was reversed) and fall in the centres of corresponding ventricular responses.

The mechanism is illustrated by the diagram below the photograph; the black rectangles represent the systoles of the auricle and ventricle and the impulse is represented as starting between auricle and ventricle and spreading simultaneously to each at alternate beats, and to the ventricle only at alternate beats. The region of block during the asphyxial period clearly lies immediately to the auricular side of the focus which creates the heart's impulses. Time in fifths of a second.

from auricle to ventricle is prolonged or ventricular responses are missed, the obstruction is at the *A-V* node (Fig. 122) or at its junction with the auricle. It is true of asphyxia, as it is true of vagus stimulation, that aberrant forms of ventricular complex appear from time to time; but this does not affect the chief conclusion. The vagus and those poisons which have been sufficiently investigated produce heart-block, not by a generalised action on the ventricle, though the whole organ is exposed to their influence, but by a selective action upon the special tissues; the intense action is upon the upper extremity of the *A-V* node.

This conclusion is confirmed by studying complete heart-block resulting from the more intensive action of the same causes, for even when dissociation of auricle and ventricle is thus promoted, there are many reasons for believing that the peripheral sections of the distributing tracts are still competent to conduct and do conduct (see page 198).

To sum up, heart-block may be produced experimentally in one of three ways. First, it may be produced by injuring the main stems of the conducting tract, or by depressing the function of these by cooling. Secondly, it arises when vagal tone is greatly increased, the auricular rate being maintained or not greatly slowed. Thirdly, it may be produced by the injection of poisons; some of these act through the vagus, others act, as do the products of asphyxia, directly on the muscle—not on the muscle of the ventricle, but again upon the *A-V* node.

The general conclusion is justified that experimental heart-block, whether produced by injury or by disturbed innervation or by poisons, is produced *by a change in the conducting power of the main neuro-muscular tract* which unites the auricle and ventricle, meaning by the main tract, the node and the bundle proper with its right and left divisions; it appears to be produced in this manner only.

Chapter XIV.

CLINICAL HEART-BLOCK.

Heart-block in man was first recorded in 1875, when Galabin (*196*) reported a case of slow ventricular action (25-30 per minute) and remarked, " we have here a heart, the auricle of which sometimes contracted twice in the interval between two ventricular pulsations, and sometimes singly in the midst of a long pause instead of just before the systole of the ventricle." He based his account upon the auscultatory phenomena, and upon curves taken from the heart's apex. Two excellent tracings which he published show beyond question that he was dealing with what we now recognise as complete heart-block. These observations are the more noteworthy because they were published the year before those of Romanes in the *Philosophical Transactions*, and several years before the work upon the cold-blooded heart had begun.

In 1885, Chauveau (*47*) described a case of heart affection and was able to recognise, in tracings taken from the radial pulse, the apex beat and the neck, that auricle and ventricle were beating at different and entirely independent rates. He compared the phenomenon to the dissociation obtained upon stimulation of the vagus.

In the year 1899, Wenckebach (*756*) and His (*319*) both described heart-block in the human subject and suggested a lesion of the auriculo-ventricular bundle as its cause. The former based his diagnosis entirely upon the arrhythmia produced in the ventricle ; the latter published clear polygraphic curves. The early publications of Mackenzie (*502*, *504*, *505*, *509*) notably advanced our knowledge, and during the last few years very many cases have been placed on record. (For collected cases, see *10*, *108*, *605*, etc..)

The first post-mortem examination showing changes in the *A-V* bundle in a case from which a clear record of heart-block had been obtained during life was published by Hay (*241*) in 1905.

The signs and grades of heart-block in man.

Heart-block in the human subject may be demonstrated by any of those clinical methods which record the activities of auricle and ventricle. It is elegantly displayed by the galvanometric method, with almost equal clearness by the polygraphic method and less distinctly by subsidiary methods.

Prolongation of the "As-Vs" interval.—Where the defect is slight it is shown by prolongation of the *As-Vs* interval, the *a-c* interval in the venous curve and the *P-R* interval in the electrocardiogram being of increased duration (*444*, *462*). The natural *a-c* interval varies between 0·1 and 0·2 second. the natural *P-R* interval between 0·12 and 0·17 second. In heart-block these intervals may be much longer. An example of continuous prolongation of these intervals to approximately 0·5 seconds is shown in Fig. 123. In Thayer's case (*723*), the *a-c* interval was increased for long

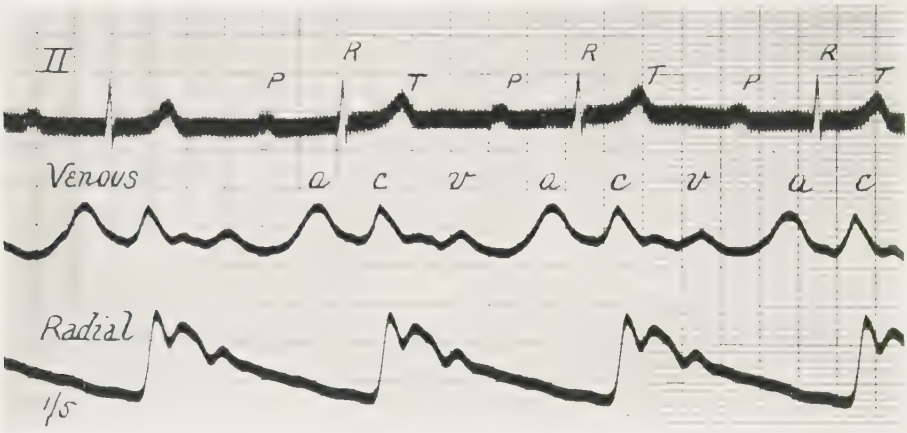


Fig. 123. Simultaneous electrocardiographic, venous and radial curves from a patient exhibiting an early grade of heart-block. The *a-c* intervals and the *P-R* intervals are approximately 0·45 seconds in duration. Time in fifths of a second.

periods to almost the full second ; the *P-R* interval to 0·7 second and more. I was fortunate in seeing Dr. Thayer's original curves, and no doubt remains in my mind that in his patient the response of the ventricle to the auricle was delayed to this extent.

When the heart rate is rapid, or prolongation of the *A-Vs* interval is great and the heart rate normal, the auricular systoles may coincide with ventricular systoles of the preceding heart cycles. In such cases *P* falls upon the preceding *T* in the electrocardiogram (Fig. 131, page 176) and *a* of the venous curve falls during the confines of ventricular systole and is exaggerated in height. Interesting examples of this kind have been published by Wardrop Griffith (*234*).

Sometimes when the *As-Vs* interval is prolonged, the beat of the auricle becomes audible and produces a form of reduplicated first heart sound or, falling near the end of the preceding ventricular systole, produces a reduplicated second heart sound (*196*, *461*).

Prolongation of the interval is also to be seen sometimes in curves from the apex beat, an observation first made by Galabin (*196*) ; in these curves and in those taken by a sound introduced into the œsophagus (*see 349*), the left auricle is responsible for the auricular summits.

Dropped beats.—When the grade of block is higher, the ventricle fails to respond to occasional auricular impulses. This form of heart-block is rarely a simple phenomenon; it is usually associated with variations in the lengths of *As-Vs* intervals over the period of the disturbance. The relation of chamber contractions may be studied in Fig. 124.

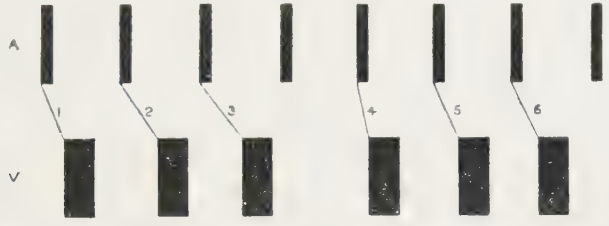


Fig. 124. The stage of heart-block, to which the term "dropped beats" is applied. Up to the point where the chief disturbance occurs, the gaps between the auricular and corresponding ventricular contractions widen out. The impulses travel to the ventricle with increasing difficulty. The fourth auricular contraction stands isolated, it yields no response; a ventricular contraction is "dropped." Following the ventricular pause, the *As-Vs* interval is short, for the tissues have rested, but it again widens as successive cycles follow.

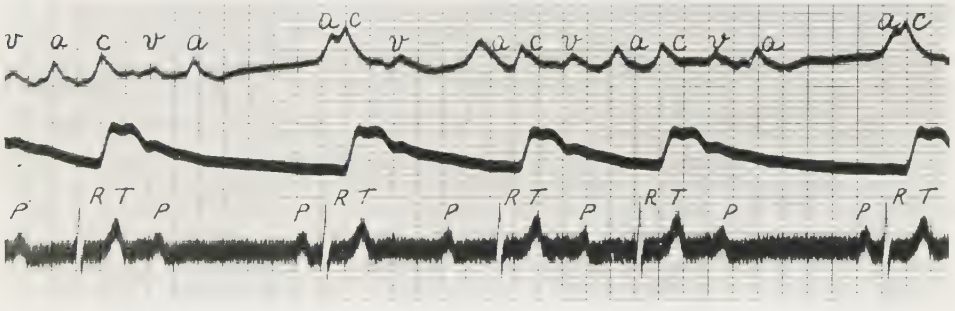


Fig. 125. Simultaneous venous, radial, and electrocardiographic curves from a patient showing the condition described as "dropped beats." After each long pause in the action of the ventricle, the *a-c* and *P-R* intervals are short; in the succeeding cycles they lengthen, while the pulse rate increases; eventually response to the auricle fails again and the events are repeated. Time in fifths of a second.

A "dropped beat" produces a pause of exceptional length and this pause breaks the natural rhythm of the ventricle. Where there is no associated variation in the *As-Vs* intervals, the length of the pause is necessarily equal to that of two regular pulse beats. But this is rarely the case; the "dropped beat" is foreshadowed by a progressive increase of the preceding *As-Vs* intervals (*cp.* Fig. 124 and 125). Moreover, the *As-Vs* interval which follows the dropping of the beat is generally curtailed (Fig. 124 and 125). These two events shorten the long pause and consequently diminish the disturbance of the ventricular rhythm. The exact

manner in which the changes happen requires closer study. Consider the first three *As-Vs* intervals of Fig. 124; as illustrated by the obliquity of the lines, the interval gradually widens, but it widens in a peculiar manner. *The increase of the second interval over the first is greater than the increase of the third over the second.* The result is a decrease of the interventricular period directly preceding the ventricular silence. *The ventricle quickens to the point of disturbance.* The shortening of the *As-Vs* interval following the pause, and the subsequent prolongation of it, produces *a similar quickening of the ventricle after the disturbance.* These relations are well displayed in the accompanying electrocardiograms (Fig. 125 and 133); they are equally well shown in many polygraphic curves (Fig. 127 and 129). In venous curves, where *a* falls further back upon the preceding ventricular waves, a characteristic and gradual heightening of the *a* wave is seen (Fig. 129).*

In some patients ventricular beats are dropped without there being an accompanying variation or prolongation of the *As-Vs* interval (*242, 761*); or there may be a suddenly developed complete block without the usual preliminary and slighter defects of conduction (see Chapter XXXI). The suggestion that block in these circumstances is due to depressed condition of the ventricle, and that that organ refuses to respond to normal impulses conveyed to it, is one that cannot be accepted unreservedly.†

Frequent failure to respond.—When there are repeated failures in the ventricular response, a simple ratio between auricular and ventricular rate becomes established. By far the commonest of these ratios is 2 : 1 block, where the auricle beats twice as frequently as the ventricle (Fig. 126, 128 and 134). In human curves of this kind, the auricular contraction immediately following the ventricular is often a little or even conspicuously premature‡, a disturbance of the dominant rhythm for which there is no present explanation, for it is the rule in partial heart-block to see no disturbance (see Chapter X).

The 3 : 1 ratio may also be seen, but it is rare; so also is the 4 : 1 ratio. More commonly the ratio is one in which 1 : 1 and 2 : 1 (Fig. 129), or 2 : 1 and 3 : 1 cycles alternate.

Complete heart-block.—The final grade of heart-block is reached when no impulses are transmitted to the ventricle. When this happens, the ventricle, having lost the controlling influence of the auricle, beats in response to a slow and regular series of impulses which it forms in its own substance. In this

* In instances of this kind the *a-c* interval may increase to as much as 0·8 seconds (*800*). The *P-R* interval to 0·6 seconds (*462, 723*) (Fig. 133), or even more.

† Especially so as well defined changes in the bundle have been found in such cases (see case reported by Cohn and Lewis (*69*)), and in others complete block has subsequently developed.

‡ For examples in venous curves see *236*; in electrocardiograms the premature auricular complexes are natural, the beats therefore are probably not extrasystolic.

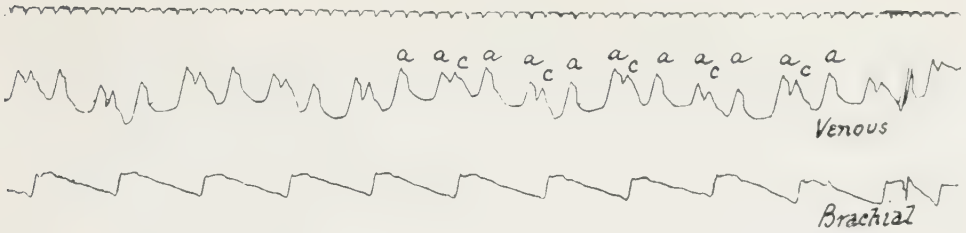


Fig. 126. Venous and arterial curves showing 2 : 1 heart-block in a patient. All the auricular contractions fall in ventricular diastole; consequently the *a* waves do not materially vary in height.

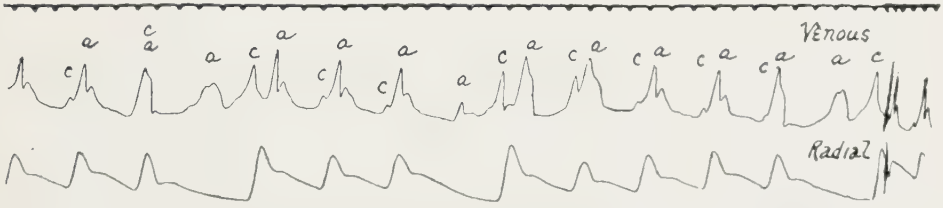


Fig. 127. Venous and arterial curves, showing prolongation of the *a-c* interval and several dropped beats in a patient. With three exceptions (the 3rd, 7th and 13th of the curve) the auricular contractions coincide with preceding ventricular contractions; *a* falls back until it coincides with *v* or *c* and is consequently exaggerated in height.

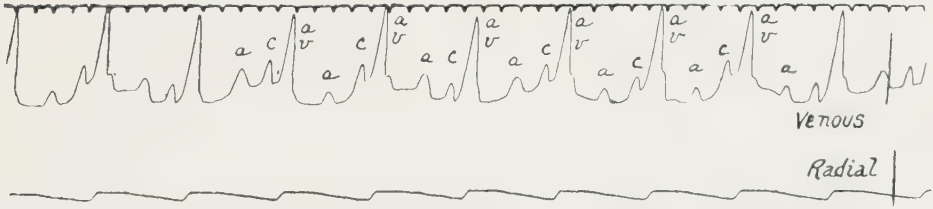


Fig. 128. Venous and arterial curves showing 2 : 1 heart-block in a young woman. The auricular contractions to which there are no responses fall with the preceding ventricular contractions and produce very tall $\frac{a}{v}$ waves. From the same case as Fig. 129.

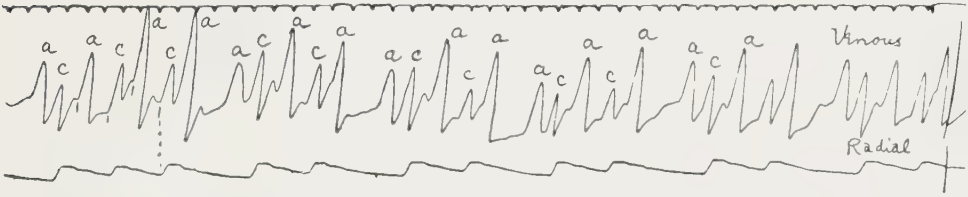


Fig. 129. Venous and radial curves from the same patient, showing frequent dropped beats, and 1 : 1, 2 : 1, heart-block. Notice the increase in the amplitude of the *a* wave as the auricular systole falls further back into the preceding ventricular systole.

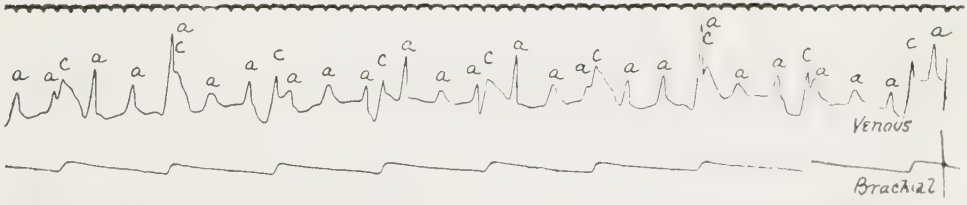


Fig. 130. Venous and arterial curves exhibiting complete heart-block in a patient. The rhythms of auricle and ventricle are regular but independent of each other. Where *a* and *c* coincide, a conspicuous summit appears.

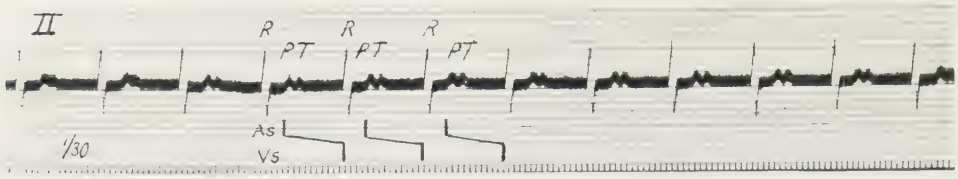


Fig. 131. An electrocardiogram showing prolongation of the P-R interval in a girl. P falls before the termination of T of the preceding ventricular cycle. This is known by comparing this curve with the following, which is from the same patient. Time in thirtieths of a second.

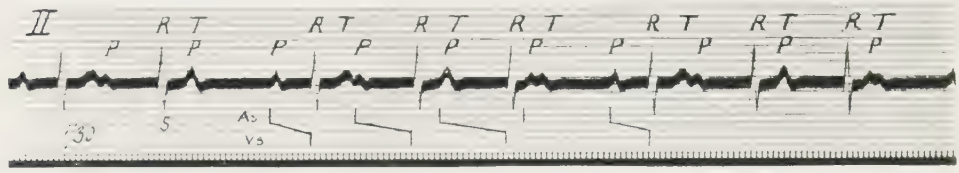


Fig. 132. Curve showing prolongation of the P-R interval and a failure of response to each third or fourth auricular impulse. Note the decreasing length of the ventricle cycles as the pause is approached; and the relation of P^7 to T^5 and compare the latter with the relation of P and T in Fig. 131. Time in thirtieths of a second.

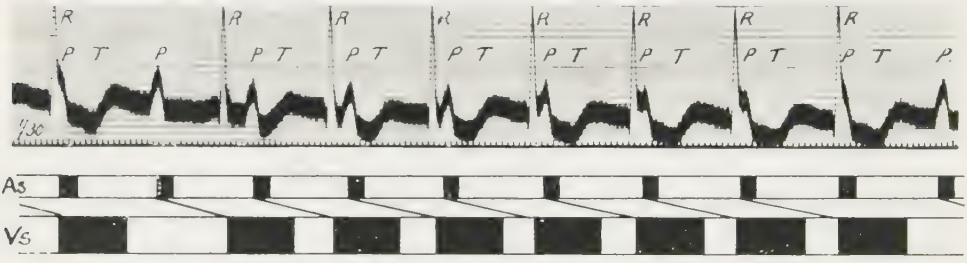


Fig. 133. ($\times \frac{4}{5}$.) Clinical curve showing very gradual prolongation of the P-R interval and two dropped beats. P falls back further and further upon T until it is actually buried in the preceding R (P^9). The preceding P-R interval (P^8) measures 0·57 seconds. Time in thirtieths of a second.

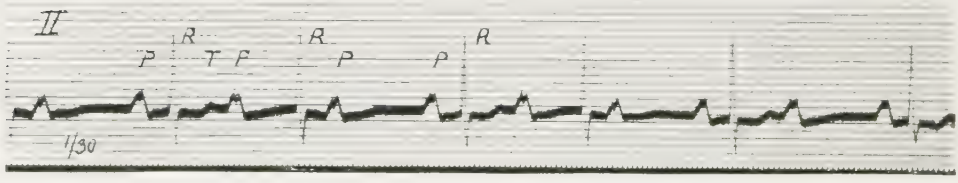


Fig. 134. Clinical electrocardiogram showing a period of 1 : 1, 2 : 1 block, passing into 2 : 1 block. Time in thirtieths of a second.

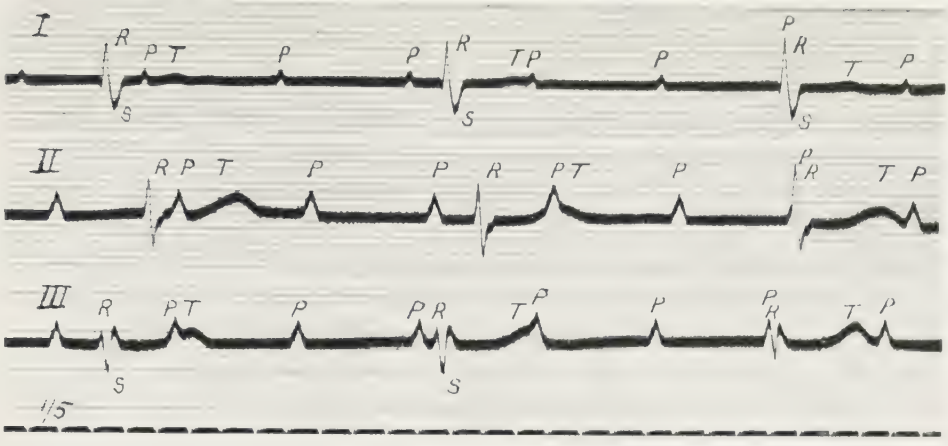


Fig. 135. Curves from the three leads in a case of complete heart-block. Time in fifths of a second.

condition, also termed "dissociation," two entirely separate rhythms are generated in the heart; the one starts in the natural pacemaker and controls the auricle, the other starts in and controls the ventricle: the former has a usual rate of 72 to the minute or thereabouts, the latter an approximate rate of 30 to the minute.

The rhythms are each regular* and quite independent; the systoles of auricle and ventricle fall with varying time-relations to each other (Figs. 130

* Not infrequently, in clinical examples, a disturbance of the auricular rhythm is manifested. As Wilson and Robinson (*784*) express it, the inter-auricular period during which the ventricular systole falls is shorter than those which follow it. This irregularity of the auricle is shown slightly, but quite definitely, in Fig. 136. The similar phenomenon referred to in describing 2 : 1 block is often more striking in degree. It is certain that the systole of the ventricle may influence impulse formation in the auricle, though the nature of the influence is unknown. To explain variation in the length of the inter-auricular periods in complete heart-block Wilson and Robinson cite the view adopted by Erlanger and Blackman (*148*), namely, that vagal tone is increased with each arterial pulse. But they point out that in rare cases the first auricular systole to follow the beginning of ventricular systole (the systole of the auricle which is superimposed on that of the ventricle) is ectopic. That is so in the case which illustrates their paper and a similar event has been reported by Cohn and Fraser (*65*) in a case of complete and incomplete block. In such instances a direct mechanical stimulation of the auricle by the ventricular systole has been suggested as the cause.

These curious influences of ventricular systole upon the auricle deserve more extended study: sometimes, in cases of actual complete block, the control of one auricular systole by each ventricular cycle influences the time-relation of all the auricular systoles to the ventricular systoles (for the second auricular systole is controlled by the first) and gives rise to a fictitious appearance of partial block.

In considering the influences of ventricular systoles upon auricular impulse formation, the reader should also refer to some curious examples published by White and myself (*490*) (especially to Fig. 18, 19 and 20 of our paper).

and 135). Isolated auricular systoles are seen in the long diastoles. In electrocardiograms of complete heart-block the super-imposition of auricular and ventricular complexes is peculiarly clear (Fig. 109, page 159 and Fig. 135). The same events are evident in venous curves, except that when the auricular contraction falls within the confines of ventricular systole the veins of the neck expand to an exceptional extent (Fig. 130). In dissociation the auricular sounds are often audible (*172*, *196*, *227*, etc.) during the long diastoles (Fig. 137), and when auricular contractions coincide with ventricular ones, intensification of the first heart sound (*234*), or reduplication of the same sound or of the second sound (*474*), is frequently to be heard. In mitral stenosis a murmur may appear at each beat of the auricle which falls during ventricular diastole, an observation (*54*, *236*) which has been made both in 2 : 1 and in complete block. The independent movements of the auricle may be seen in some cases upon a fluorescent screen (*43*, *641*) ; they may be recorded at the apex beat.

The pulsations of the auricles are also conducted along the arteries (*102*, *180*, *504*), and not infrequently appear upon radial tracings (Fig. 136). The mode of transmission to so distant a point is not clearly understood, but it is probable that the base of the aorta acts in much the same way as does an œsophageal sound, such as is used for recording the movements of the left auricle, and that the contractions of the auricular part of the heart which is wrapped around the aorta produce changes in systemic arterial pressure which are transmitted to a distance.

The causes of clinical heart-block.

When we inquire into the causes of clinical heart-block, two conclusions should stand out prominently in our minds ; (1) conduction between auricle and ventricle depends, as experiment has clearly taught us, upon the auriculo-ventricular bundle and upon this alone ; (2) all the forms of experimental heart-block which have been sufficiently investigated in the mammal have been shown to result from defects in this tract of tissue. These two conclusions are based upon relatively simple though exacting methods of investigation ; disease undertakes few simple experiments, its lesions are rarely localised, usually the damage is widespread. Our first conclusions apply to the lower orders of mammalia ; we may expect morbid anatomy to demonstrate in man, what has been discovered in the lower animals; yet morbid anatomy is still relatively an inexact science and we cannot expect its evidence to be so clear or so convincing as is experimental evidence. There is a further consideration ; the facts harvested in the laboratory are gathered by those methodically trained as investigators ; the opportunity of recording the effects of the disease, that is the relation of lesions of the *A-V* system to heart-block in the human heart, is offered to many who are inexperienced in precise methods both of recording the heart beat and of examining the heart microscopically. Thus, when we reflect, we shall be

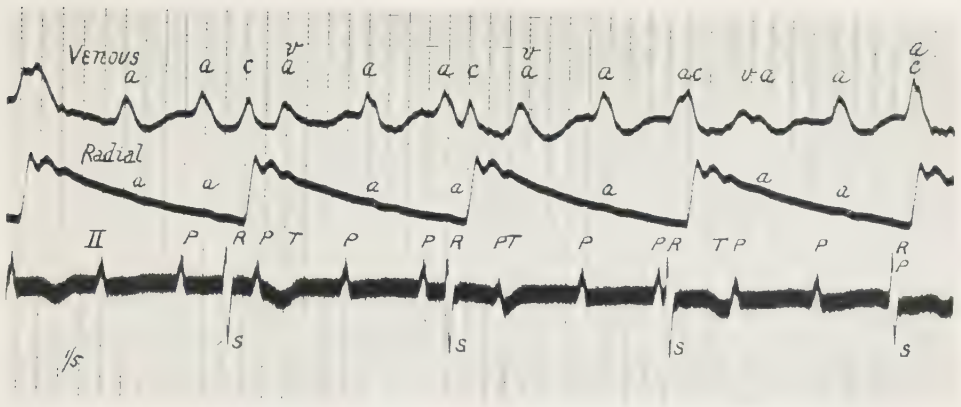


Fig. 136. ($\times \frac{4}{5}$.) Simultaneous venous, radial and electrocardiographic curves in a case of dissociation. In the venous curve, frequent *a* waves and occasional *c* and *v* waves register the movements of auricle and ventricle; the last is a composite *a* and *c* wave. The radial curve is of large amplitude and minute *a* waves are quite clearly inscribed upon it. The constant time-relation between these little waves, the *a* waves in the jugular and the *P* summits of the electrocardiogram, should be observed. Time in fifths of a second.

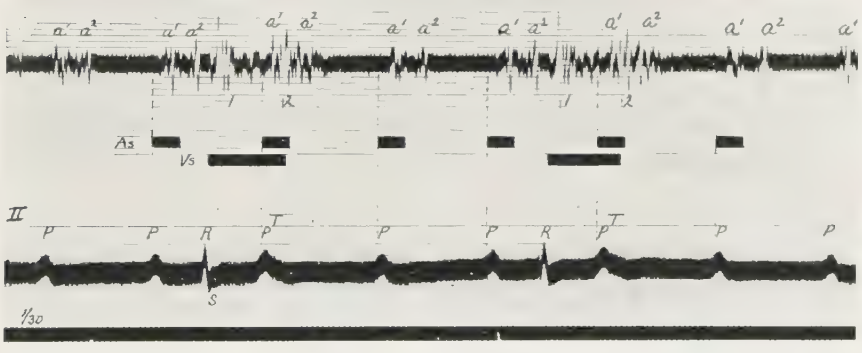


Fig. 137. ($\times \frac{4}{5}$.) Simultaneous electrocardiogram and heart sound record, the latter taken from the apex beat. From a case of complete heart-block. The sounds of the auricle are double (a^1 and a^2) and occur a little after the beginning of each *P* summit. The reason of the double sound, which is unusual, is discussed in the original description (*474*). Time in thirtieths of a second.

surprised, not that reports upon the human subject are sometimes in conflict with the testimony of experiment, but that, viewed generally, they are in such remarkable harmony with them and with each other. A mass of human material has been studied, some of it has been suitable for this purpose, much of it has been quite unsuitable; it can scarcely be questioned that a large proportion of the reports are inadmissible, marred as they are by unavoidable

or avoidable imperfections. The task of the present generation, and of those who follow, would have been lighter had those reports been suppressed which did not contain simple and less questionable data. I emphasise these points of view, because a general survey must take account of them.

Heart-block arises in the human subject from the same causes as in experiment.

1. *From lesions of the conducting tract.*—When persistent heart-block of a high grade has been demonstrated during life, lesions are usually found affecting either the *A-V* node or the bundle; scores of cases* might be cited in support of this statement. As a general rule the lesions are prominent (*11*, *37*). On the other hand, no instance of complete destruction of the bundle has been satisfactorily demonstrated, where impulses were known to have passed from auricle to ventricle a reasonably short time before death. The most exquisite example of a lesion created by disease is that of the child fully reported by Armstrong and Mönckeberg (*8*); complete heart-block resulted in this patient from an endothelioma arising in, confined to, but totally destroying the hinder part of the *A-V* node. A single case, often cited as an exception, is that of Heineke, Müller and Hösslein (*248*). Partial heart-block was recorded and *six weeks* later complete destruction of the bundle by fibrosis, hæmorrhage and calcification is said to have been found; the hæmorrhage certainly, the calcification very possibly, was of terminal origin.† But it is clear from reliable records that, excepting instances of complete transection, it is not possible to estimate microscopically the antecedent grade of functional impairment. There are cases on record in which gross lesions have been described, but where only the slighter grades of heart-block or merely the temporary defects of conduction have been observed (*69*, *220*, *228*). There are cases in which, with far slighter grades of apparent damage, dissociation has been complete and permanent (*398*). Further, slight changes in the conducting system are common in elderly subjects (*37*), though heart-block is comparatively rare; they were found in 70 per cent. of all cases of heart affection examined by Sternberg (*707*) in a search through 72 hearts.

When in instances of gross damage, a few fibres appear to be intact, it is impossible by examining them to estimate their previous functional efficiency; and we know that undamaged fibres in small numbers are sufficient to serve as normal conductors. In examples of diffuse but slighter change, the functional capacity of the bundle as a whole is equally difficult to gauge when it is

* A complete list and analysis of cases in which cases of heart-block have come to post-mortem examination up to the year 1910 was published in my book (*447*, *page 100*).

† Holtz and Krohn's case (*332*) should not be cited, for neither the account of the microscopic anatomy, nor the interpretation of the curves is satisfactory. In Martin and Klotz's case (*541*), no curves were published, and there was no proof of conduction, neither can it be reasonably inferred.

submitted to microscopy. The seeming discord between the grade of heart-block and the degree of tissue change is to be explained in some cases by the insufficiency of our histological methods,* in others by change in the lesion in the interval between the times when the functions of the system on the one hand and the structure of the tissues on the other were investigated.

2. *The vagus and clinical heart-block.*—That vagal impulses may produce heart-block in healthy human subjects, has been shown. If the nerve be pressed upon in the neck, prolongation of the *P-R* interval is witnessed (*653*); the reaction is abolished by atropinisation. Higher grades up to dissociation have been produced in heart cases by the same procedure (*654*).† Heart-block can be induced through the vagus reflexly also by pressure on the eyeball; excellent examples (controlled by atropine injections) have been published by Petzetakis (*592*). The effect of vagal compression is greater when a tendency to heart-block is pre-existent. Thus, when prolonged *As-Vs* intervals have been induced by the administration of digitalis, vagal compression may prevent the response of the ventricle for many auricular cycles (*625*). One of the most notable examples of heart-block arising from vagal stimulation is that published by Mackenzie (*509*); he observed that swallowing, an act which provokes an inhibitory cardiac reflex, produced a failure of ventricular responses in a patient, who previously exhibited a prolonged *a-c* interval.‡ A curve from this patient is reproduced in Fig. 138.

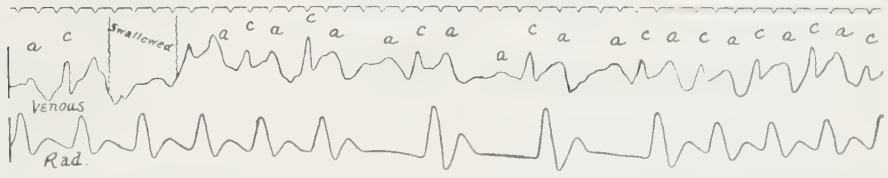


Fig. 138. (*Mackenzie, Brit. med. Journ., 1906, II, 1110.*) A polygraphic tracing from a case of partial heart-block (prolonged *a-c* interval), showing the effect of swallowing upon the heart's mechanism. After an interval of three heart-cycles, temporary 2 : 1 heart-block is produced. A clinical example of an increase in the degree of heart-block as a result of vagal inhibition.

Heart-block conditioned in man by these interferences is necessarily temporary; it is not possible to maintain heart-block of uniform grade for very long periods in experiment by vagal stimulation. It is open to question, therefore, if persistent heart-block in the human subject can ever

* Before the discovery of Wallerian degeneration, the reason for many defects of the nervous system was unrecognised.

† The conclusion that the left nerve has a more marked effect on conduction does not seem to me justified as yet.

‡ A similar instance has been reported by Laslett (*412*).

be attributed to nervous influence. Unquestionably, in most patients, the persistent heart-block of high grade is not of vagal origin for it is unaffected by full doses of atropine. Atropine usually has no effect on the ventricular rate in cases of dissociation. So far as I am aware this drug has never been known completely* to abolish heart-block unless such heart-block has been induced by the administration of drugs. That it may reduce the grade of a digitalis block or abolish it altogether is known (*517*, *625*, *743*).

It may be that the vagus is accountable for transient heart-block in man. It may infrequently be responsible for temporary increases in the degree of block through reflex channels. It may be responsible for continued block when the vagal mechanism is stimulated by drugs. Nevertheless, it has not been shown that persistent heart-block of high grade is due to this cause; our evidence is decidedly opposed to this conclusion, which has been formed with more persistence than wisdom.

3. *Heart-block as a result of poisoning.*—It has been shown (*646*) that heart-block occurs in children dying from anterior-poliomyelitis. The clinical details of the cases, and the curves, seem to establish this form of heart-block as asphyxial.

The most remarkable instances of toxic heart-block in man are those following the administration of drugs of the digitalis group. Until recently it has been thought that conduction changes cannot be induced in the heart of man by therapeutic doses of digitalis except in cases where there is already a predisposition to this defect. But it has now been shown that normal subjects also exhibit the reaction (*62*, *66*, *708*, *769*). We are indebted to Mackenzie, (*504*, *505*, *517*), for the first observations, and for the greater part of our knowledge of digitalis effects. He has shown that where there is a slight defect of conduction, that defect can readily be increased and rendered more prominent—by full doses of digitalis. This conclusion has been abundantly confirmed by other workers (*95*, *306*). In such patients, high grades of block, even dissociation, may develop. Similar effects are observed with strophanthus and squills (*788*). Digitalis heart-block seems in some patients to be of vagal origin, for it may be abolished by full doses of atropine; in other cases its action appears to be directly upon the junctional tissues (*106*, *517*).

* An observation of Barringer (*22*) upon a case of temporary dissociation seems to demonstrate that the heart-block was in a measure vagal in origin, but his curves do not demonstrate that it arose entirely from this cause.

NOTE ON DISSOCIATION OF THE TWO AURICLES.

In 1906 Wenckebach (*760*) figured and described a series of curious curves which he interpreted as resulting from dissociation of the two auricles. Wenckebach's explanation is inacceptable to-day in that such dissociation is unknown in experiment and in that it is opposed to the now widely supported view, that physiologically the muscle of the two auricles is to be regarded as one undivided mass. But an alternative explanation of Wenckebach's curves still fails us; no similar mechanism has since been described to my knowledge. The importance of the curves lies perhaps in their strangeness.

NOTE ON DISSOCIATION OF THE TWO VENTRICLES (HEMISYSTOLE).

From time to time interpretations of clinical curves have been put forward in which independent contractions of the ventricles are supposed to be evidenced. For the most part these curves have been polygraphic. Notable examples of such curves are those published by Mackenzie (*501*, Fig. 309, 310, 313 and 314 of that book), and those published by Hewlett (*308*). Mackenzie's curve, Fig. 309, almost certainly represents successive extrasystoles, his Fig. 310 and the curves published by Hewlett would now be regarded generally, I think, as exhibiting unusually large "stasis" and *b* waves in the long diastoles of the venous curves. Mackenzie's Figs. 313 and 314 still lack an explanation. The same idea of hemisystole was used in explaining the electrocardiographic curves of ventricular extrasystole by Kraus and Nicolai (*391*), but this explanation is now recognised as a mistaken one. There is, in fact, no clinical evidence of "hemisystole"; there is nothing to make us believe that one human ventricle may beat without the other, continuously or occasionally. A continuous beating of one ventricle while the other stands quiescent would be a condition clearly incompatible with a continued circulation. Even in the dying heart it is most difficult to conceive of an occasional beat confined strictly to one ventricle, for the two chambers have a continuous musculature and in large measure a common blood supply.

The question of hemisystole has been contested on the basis of experimental work. Opinion is decidedly against its occurrence. In the dying heart one ventricle may beat while on superficial inspection the other chamber may appear still, but closer inspection or a finer method of recording demonstrates movement in the second chamber also. While it cannot be said at present that there is any real foundation for the hypothesis, it can be said that our knowledge of the anatomy and physiology of the ventricles is strongly opposed to it.

The following additional papers, which deal with this question at greater length, may be consulted (*251*, *252*, *604*, *606* and *623*).

CHAPTER XV.

NEW RHYTHM CENTRES (ATRIO-VENTRICULAR RHYTHM).

In 1903, Engelmann (*135*), while recording the movements of auricle and ventricle in the frog's heart, noticed in certain animals that when the region of the sinus is separated from the rest of the heart by tying (1st Stannius ligature) the auricles and ventricles subsequently beat simultaneously. He attributed this newly developed rhythm to impulses arising in the *A-V* ring. A year later a similar mechanism following vagal stimulation in the tortoise and rabbit was described (*492*), and attributed to a similar cause. Since the discovery of the *A-V* node, the impulses yielding simultaneous systole of auricle and ventricle have by general consent been supposed to arise in this structure; the suggestion first came from the papers of Hering and Rihl (*303*), and Mackenzie (*512*, *513*), describing certain isolated and premature beats discovered in patients.*

It has been shown that atrio-ventricular rhythm may be induced in the mammalian heart by a number of different procedures. The most constant and striking method is by destroying the *S-A* node or by cooling the same structure (*71*, *204*, *493*). It is also to be produced by warming the *A-V* node. Atrio-ventricular rhythm may also follow interference with the nerves of the heart, especially combined stimulation of the right vagus and left sympathetic nerves (*665*).

The change in the heart's mechanism is well illustrated by experiments in which the region of the *S-A* node is cooled. A leaden tube is laid along the sulcus terminalis of the auricle, and iced water is passed through it. The heart rate is retarded almost immediately and its action changes (Fig. 139). The *P* summits of the natural electrocardiogram disappear and simultaneous action of the auricle and ventricle becomes established. This is seen in the electro-cardiogram, but is more clearly deciphered in the muscle curves. The last three ventricular complexes of Fig. 139 are similar to the first complexes of the curve; the corresponding beats are still supraventricular in origin; the auricular complexes are of altered form and

* Mackenzie, in the same series of papers (*511* *513*), also supposed that what we now know to be auricular fibrillation arises in this node and provisionally termed this condition "nodal rhythm." This hypothesis was discarded in 1910, when I published a paper proving the nature of auricular fibrillation and recording an actual instance of nodal rhythm (*434*).

Fig. 139. (*Heart*, *1914*, *V*, *247*, *Fig. 1.*) Myocardiographic curves (*A* = auricle; *V* = ventricle) and electrocardiogram from lead *II* showing the effect of applying cold to the sulcus terminalis in a dog. With the reduction of temperature (see signal) the *S*-*A* rhythm slows; at the fifth cycle there is escape of the *A*-*V* node, though the auricle, being already in contraction, does not respond. Subsequent cycles show the fully established *A*-*V* rhythm; the auricle is represented in the electrocardiogram by a minute dip preceding the upstroke of *R*. The *A*-*V* and *P*-*R* intervals are given in decimals of a second. Time in fifths of a second.

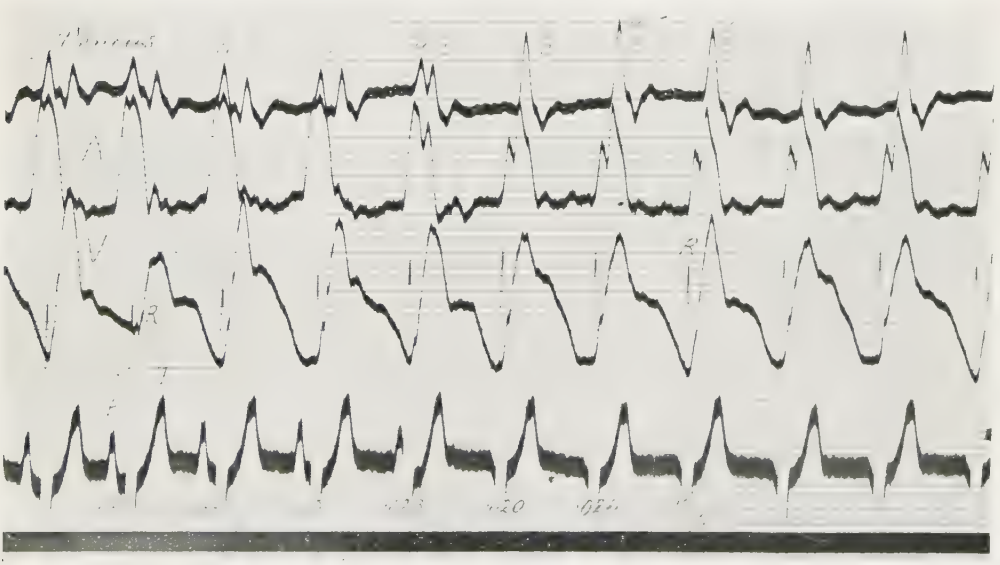


Fig. 140. (*Heart*, *1914*, *V*, *247*, *Fig. 3.*) Similar curves from another animal, showing the awakening of *A*-*V* rhythm on the application of cold. The uppermost curve is from the veins of the neck and shows the exaggerated a wave accompanying the simultaneous contractions of auricle and ventricle. The *P*-*R* intervals are given in decimals of a second. (The intervals have been entered incorrectly on this curve, they should all be moved one cycle to the right.) Time in twenty-fifths of a second.

lie almost completely buried in those of the ventricle : the initial descent of *P*, preceding *R* by an interval of 0·027 of a second, is alone visible. An example of a similar change is shewn in Fig. 140. In this figure a volume curve of the veins of the neck is added : when the auricle and ventricle beat synchronously, the normal *a* and *c* waves are replaced by a tall wave $\frac{a}{c}$; this exaggerated wave is due to the auricle forcing its contents into the veins of the neck, since during the systole the tricuspid valve is closed.

When *A-V* rhythm develops in the dog's heart, there may be, as in the present examples, a conspicuous reduction of the *As-Vs* interval from the normal of 0·08–0·10 of a second to 0·02 or 0·03 of a second. The reduction may be greater, and the interval may vanish altogether or become of opposite sign ; that is to say, the ventricle may beat a little before the auricle. On the other hand, the reduction may be less (Fig. 141). Be the reduction slight or great, the new rhythm arises in the *A-V* node according to present day conceptions. The meaning of the variations in *As-Vs* intervals, which occur not only from animal to animal, but even in the same animal from time to time (Fig. 140 and 141 are from the same animal), will be discussed presently.

Of peculiar interest is the shape of the auricular representative in *A-V* rhythm. Though inconstant in form it usually presents the inversion of Fig. 139 ; where it is buried in the corresponding ventricular complex, its form is not clearly seen, but it may be unveiled by breaking the *A-V* bundle and producing *A-V* dissociation (*547*). The auricle then responds to *A-V* nodal impulses, the ventricle responds more slowly to its inherent impulses.

The usual form of the auricular complex, when *A-V* rhythm prevails, is that of Fig. 142 ; it starts as a rapid downward deflection of considerable extent and has a slower upstroke.* Less commonly, the complex is not so extensive and consists of a few minute deflections. These variations in the form of the auricular complex are scarcely to be explained as resulting from variation in the architecture of the auricles in different animals, for they are more distinct than are variations in the normal auricular complex in those of the same species. Possibly the excitation wave, in travelling backwards in the auricle, follows a less constant and ordered route than in its forward propagation.

For one or more cycles at the transition from *S-A* to *A-V* rhythm, the auricle may respond to an *S-A* impulse, while the contractions of the ventricle are hastened by the development of *A-V* nodal impulses to which the last named chamber responds. Evidently the transitions of Fig. 139 to 141 are of this kind. Thus in the fifth cycle of Fig. 139 the auricle is represented by a complex which is like those preceding it, but the *P-R* interval is reduced because the first *A-V* impulse culminates and excites the ventricle before response to the natural impulse is due. The auricle does not respond to the

* According to Ganter and Zahn (*206*), this form of complex is always associated with an origin of the beat from the upper part of the *A-V* node ; this conclusion is, I think, questionable.

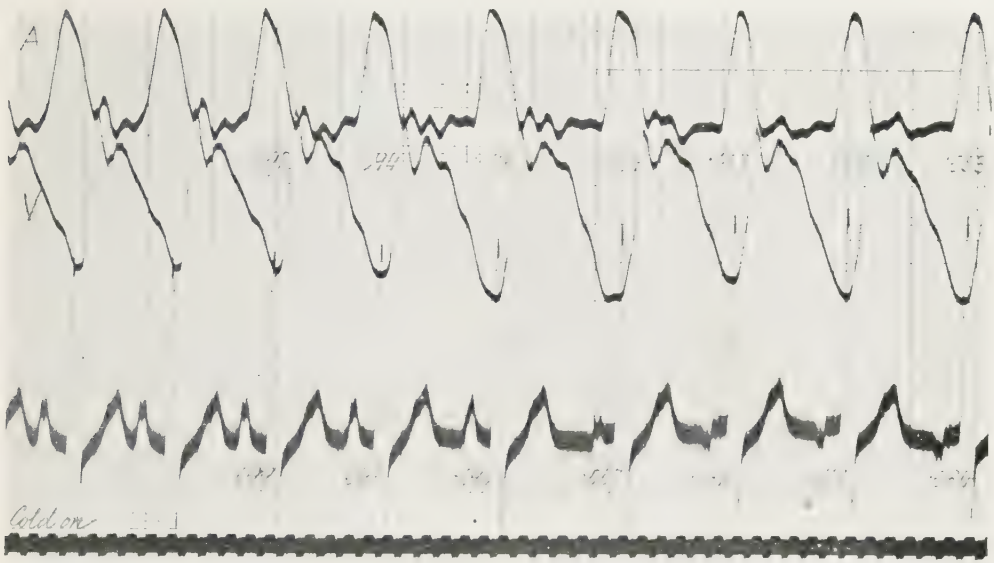


Fig. 141. (*Heart, 1914, V, 247, Fig. 2.*) Similar curves from the same animal as in Fig. 140, showing slowing of the heart and alteration of the pacemaker subsequent to the application of cold. *P* becomes inverted and the *P-R* interval is slightly reduced. Note the change in the shape of *P* at the transition. Intervals in decimals of a second. Time in twenty-fifths of a second.

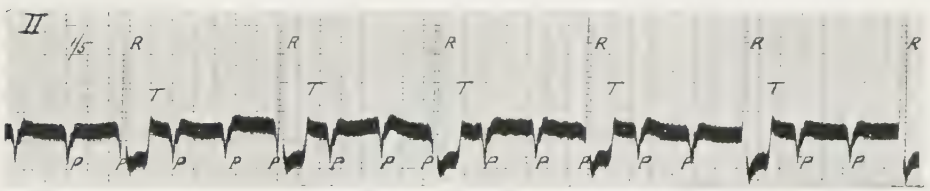


Fig. 142. (*Meakins, Heart, 1914-15, V, 281, Fig. 9.*) ($\times \frac{4}{5}$.) An electrocardiogram from lead *II* in a dog. *A-V* rhythm was induced by cooling the *S-A* node, and during the maintenance of this rhythm the *A-V* bundle was broken by clamping. In the curve dissociation is shown between the auricular and ventricular contractions, and the unusual form of the auricular complexes is plainly to be seen. Time in fifths of a second.

A-V nodal impulse because when this impulse arrives the auricle is already in contraction and consequently refractory. A transition of a somewhat different and more complex type is shown in Fig. 141. Here the first altered auricular complex (the sixth in the curve) is intermediate in type between that which precedes and succeeds it. In instances of this kind it is probable that the *S-A* and *A-V* impulses entered the auricular substance at the same moment and that for one cycle the response was partially to one impulse and partially to the other.

Evidence that these new rhythms arise in the A-V node.

That the *A-V* node is responsible for certain rhythms in which the auricle and ventricle contract simultaneously is now generally accepted. The node is an integral part of the system of tissue joining the two chambers, and impulses arising in the node may be supposed to spread simultaneously to these chambers. The two nodes (*S-A* and *A-V*) are alike structurally and are probably morphological homologues ; the *S-A* node is known to possess a rhythmic power in high degree, the *A-V* node is expected from its structure to possess a similar power.

The electrocardiograph tells us that the ventricular beats, while *A-V* rhythm is in progress, arise above the division of the *A-V* bundle (Fig. 139-141), for the impulses are distributed to the ventricle in normal fashion. That they are supraventricular is established by the shape of the ventricular complexes and by the following experiment. If during *A-V* rhythm the septum is cooled on the ventricular side of the node (*797*), or if the bundle is clamped (*547*), the ventricle fails in its responses while the auricle continues to beat.

The inversion of the auricular complex in *A-V* rhythm indicates an upward passage of the excitation wave as opposed to its usual downward course ; this reversal of propagation is also indicated if we lead from two direct contacts placed upon the sulcus or parallel and near to it.* When *S-A* rhythm prevails it is the upper contact which first becomes negative, during *A-V* rhythm it is the lower one, as Wybauw (*795*) first demonstrated, and as I have myself often found. But perhaps the most convincing evidence is that *A-V* rhythm is retarded by cooling,† and accelerated by heating, the region of the node (*797*).

It has also been stated (*550*) that negativity is first developed in this region of the heart while *A-V* rhythm is present.‡ The conclusion that the rhythm arises in the *A-V* node is not only supported by these observations, but it is in harmony with so many other conclusions, that it is scarcely to be questioned.

Variation in "A-V" rhythm intervals.

The conclusion that impulses which arise in the *A-V* node may be responsible for heart beats in which auricle and ventricle beat exactly simultaneously, or for heart beats in which auricle or ventricle has a short

* This method, though its results are not often misleading when the two contacts are in the immediate neighbourhood of the rhythm-producing centre, or in the direct line along which the excitation wave is propagated, is nevertheless open to some criticism. The curves are confused by extrinsic deflections.

† Zahn's statement that he cools particular and known regions of the node is scarcely convincing, but there seems no reason to doubt the more general conclusion.

‡ This evidence is open to criticism, for it is not possible to lay an electrode directly upon the *A-V* node, and even though it can be placed inside the heart near the node, the precise region examined is difficult to ascertain subsequently.

lead, was first reached by considering the time intervals which separate the contractions of auricle and ventricle. We know that conduction in the auricle is rapid, we know that conduction in the Purkinje substance of the ventricle is still more rapid ; considering a normal *As-Vs* interval of 0·10 of a second, some 0·03 of a second may be allowed for conduction through the auricle, a smaller interval may be allowed for conduction from bundle to ventricle. Somewhere in the path of conduction there is an unexplained delay of from 0·04 or 0·05 of a second. It is generally accepted that this delay occurs in the fine fibres of the *A-V* node ; in Chapter VII the reasons for believing that slowness of conduction is associated with smallness of fibre have been given. One worker believes that he has demonstrated delay in this structure by direct experiment (*286*), but the reasons for his conclusion cannot be accepted unreservedly. We have no proof, yet we may assume, that the chief delay is in this structure. Thus it has been concluded (Chapter XIII) that it is the depressed function of this node which exaggerates the delay in *A-V* block, caused by asphyxia or vagal stimulation. It is to be inferred that the point at which the natural delay occurs is also that at which the delay is most susceptible to exaggeration. The strongest ground for our hypothesis is that it harmonises with many distinct observations, and is, so far as I know, discordant with none. It is further argued that the variations in length of the *As-Vs* or *Vs-As* interval as the case may be, is due to variations in the level of the node from which the impulses arise ; and for this last assumption there are a number of arguments and some direct evidence. If the *As-Vs* interval is but slightly reduced, the portion of the node towards the auricle is considered the rhythm centre (Fig. 141) ; if the beat of the auricle and ventricle is simultaneous (Fig. 140) or a *Vs-As* interval is developed, then the portion of the node towards the ventricle is said to be active. A powerful argument in favour of this thesis is supplied by those agencies which produce disturbances of *A-V* conduction by an action concentrated upon the region of the node. Thus if *A-V* rhythm prevails and the reduction of the *As-Vs* interval is not great, vagal stimulation—sufficient in degree to produce heart-block while the heart is responding to the *S-A* node—produces the natural form of heart-block ; that is to say, the *As-Vs* interval increases and the ventricle occasionally fails to respond. But if the *As-Vs* interval is conspicuously reduced, or if a *Vs-As* prevails, then a similar stimulation of the vagus produces a *reversed* block ; the *As-Vs* interval decreases (a *Vs-As* interval increases) and *auricular* contractions are missed (*466*). To explain these phenomena I use the accompanying diagrams. In Fig. 143 the impulse is supposed to arise in the upper, in Fig. 144 in the lower, part of the node (*N*) and to spread to the auricle (*A*) and simultaneously to the ventricle (*V*) through the bundle (*B*). Vagal impulses acting on the *A-V* node cause a lowering of conductivity in this structure, and according as the obstruction is more on the auricular, or more on the ventricular, side of the rhythm centre, so the beat of the auricle or of the ventricle is delayed. As the vagal action

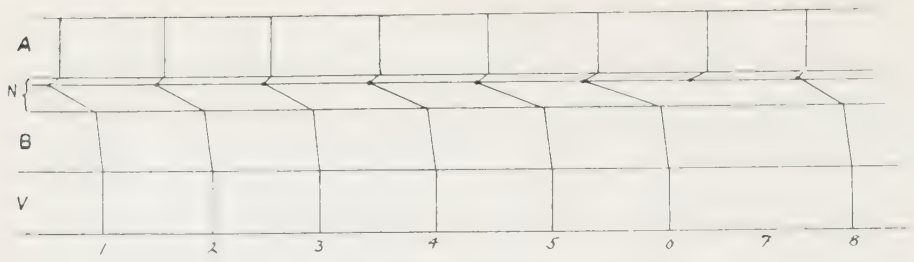


Fig. 143. A diagram illustrating the disturbances produced when, during *A-V* rhythm originating in the upper part of the node, the vagus is stimulated. The increased vagal tone produces heart-block of the usual kind; the *As-Vs* intervals widen and eventually the ventricle fails to respond.

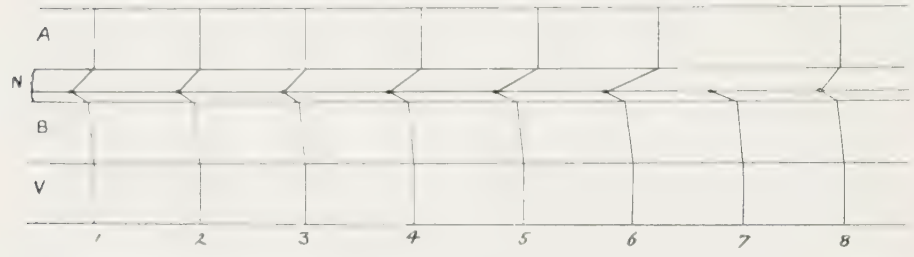


Fig. 144. A diagram illustrating the disturbances produced when, during *A-V* rhythm originating in the lower part of the node, the vagus is stimulated. The increased vagal tone produces a reversed heart-block; the *As-Vs* interval shortens (this is not, but should be, shown), a *Vs-As* interval develops, and eventually the auricle fails to respond.

in a single animal is constant, so it must be assumed that it is the position of the rhythm centre which has changed. This modified action of the vagus is not an isolated example; similar phenomena are witnessed when the changes in conduction intervals are conditioned by premature contractions (*490*). I hesitate to call up the observations of Zahn (*797*, *798*), and those of Meek and Eyster (*550*), in support of the hypothesis that rhythms are developed at different levels of the node. The former states that, when the *As-Vs* interval is slightly reduced, temperature changes over the auricular portion of the node affect the rate of the rhythm, and that, when the reduction of the *As-Vs* interval is great, temperature changes affect the ventricular portion of the node. The latter assert that the point of primary negativity lies nearer to the coronary sinus, or nearer the tricuspid valve, when the longer or shorter intervals, respectively, prevail. I hesitate because this exact location of events in a small and buried structure appears to me precarious, and also because the method of leading in Meek and Eyster's experiments is open to certain objections. For the reasons given I believe, nevertheless, that the conclusions at which they arrive are the right ones.

Influence of nerve stimulation upon "A-V" rhythm.

Many observations have been undertaken with a view to ascertain the innervation of the *A-V* node. The effects of left and right vagal stimulation upon conduction from auricle to ventricle has been dealt with in a previous chapter (page 164), but these should be distinguished from those now discussed. It is generally accepted that the right vagus has a greater retarding influence upon the *S-A* rhythm than has the left. It is by no means so clear that the left vagus has a greater retarding influence upon *A-V* rhythm than has the right. Quite exceptionally in dogs it is sufficient to stimulate the right vagus to transfer the pacemaker from the *S-A* to the *A-V* node ; much more often a similar stimulation during *A-V* rhythm causes an opposite transference. The influence of both vagi over rhythm production in the *A-V* node is powerful ; but the right nerve, in my experience, is predominant in this respect in most dogs (*466*). The left vagus seems to act more powerfully upon the *A-V* node than upon the *S-A* node in most experiments, but so does the right nerve ;* both the degree of vagal control and its distribution is variable from animal to animal. The left sympathetic nerve seems to accelerate rhythm production in the *A-V* node powerfully and to a greater extent than the right nerve (*665*) ; so when the right vagus, which holds the *S-A* rhythm in abeyance, is stimulated simultaneously with the left accelerator, *A-V* rhythm displays itself ; and this is the more explicable since the left accelerator is said to have relatively little influence upon *S-A* rhythm (*403*). Sometimes, indeed, isolated stimulation of the left sympathetic suffices to induce an *A-V* rhythm (*403*).

The reactions to stimulation are so complex and the experiences of different observers, or of the same observer in different experiments, are so variable that a further analysis of the relative influences of the right and left nerves upon the two nodes cannot here be attempted in detail. Suffice it to say that the *A-V* node is richly supplied by inhibitors and accelerators ; and that while the right vagus has an unquestionable predominance over the left nerve so far as the right or *S-A* node is concerned, the left sympathetic has a predominance over the right nerve so far as the left or *A-V* node is concerned. To this extent the nerve supply appears to be homolateral. But the crossed effects, the action of left inhibitor and left accelerator upon the *S-A* node, and of the right inhibitor and right accelerator upon the

* One of my original reasons for stating that the vagus has a greater influence upon the *A-V* node than upon the *S-A* node in certain dogs was the observation that in these vagal stimulation would convert an *A-V* rhythm (induced by cooling the *S-A* node) to a *S-A* rhythm. As Schlomovitz and his fellow workers (*689*), have justly remarked, this reasoning is inadmissible, in that the cooling of the *S-A* node depresses the influence of the vagus upon it. Nevertheless, and in view of the profound slowing effect of the vagus upon *A-V* rhythm itself, I retain my former conclusion, being unable to accept the view which these authors express that the vagus has a lessened effect on nodal tissues, as these are traced downwards through the heart in the dog. Were that the case simple vagal stimulation, especially right vagal stimulation, would usually produce *A-V* rhythm ; that is not so.

A-V node are also to be obtained, and certain of these crossed effects seem to be as powerful as those which are uncrossed.

The original papers should be consulted by those who desire fuller and more exact detail of the numerous observations which have been made upon the nerve supply of the two nodes (*6*, *207*, *302*, *403*, *466*, *550*, *665*, *667*).

Clinical examples of A-V rhythm.

Atrio-ventricular rhythm was first described and figured in man by Belski (*30*) in 1909. Shortly afterwards I published (*434*) an example of a rapid rhythm* in which auricle and ventricle contracted simultaneously,

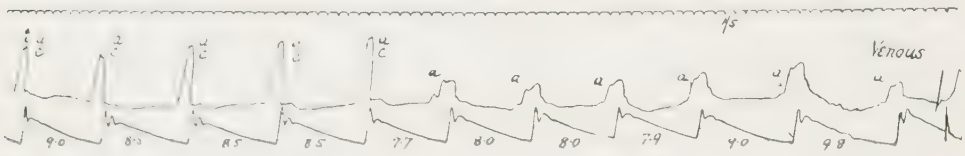


Fig. 145. An example of *A-V* rhythm, determined by polygraph in the human subject. The *A-V* rhythm prevails for five cycles, and tall waves $\frac{a}{c}$, due to simultaneous contraction of auricle and ventricle, are seen in the curve. At the sixth cycle the *S-A* rhythm escapes and controls the movements of the heart to the end of the curve. At first the heart rate is slow; rate 35 per minute; the escape of the *S-A* node has been due to a quickening of its rhythm. The lengths of the cycles are given in fifths of a second. From the same case as Fig. 146 to 148.

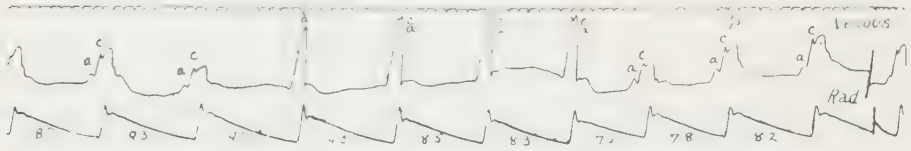


Fig. 146. A curve from the same patient showing the onset of *A-V* rhythm as the heart slows, and the re-establishment of *S-A* rhythm accompanying an increase of its rate.

and was able to present both polygraphic and electrocardiographic curves. At later dates very many examples have been placed on record (*201*, *460*, *544*, *636*, *766*, *780*, *782*). The appearances of this rhythm in polygraphic curves and in electrocardiograms are identical with those described in the experimental section of this chapter. In the venous curves the striking characteristic is the high wave produced by simultaneous contraction of auricle and ventricle. The waves are most exaggerated when the systoles of auricle and ventricle begin exactly together. This *A-V* rhythm in patients discloses itself when the *S-A* rhythm is depressed. In

* A rhythm which should not now be termed atrio-ventricular rhythm, but atrio-ventricular tachycardia.

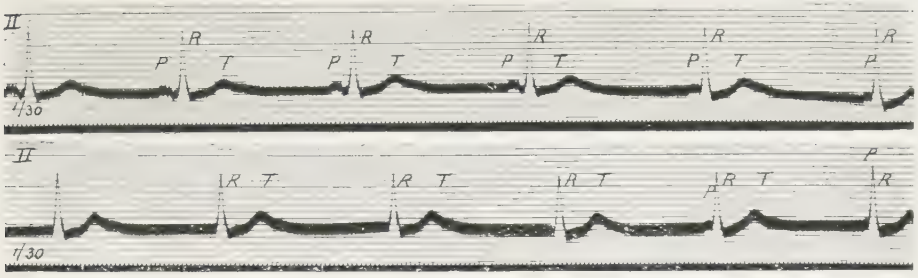


Fig. 147 and 148. ($\times \frac{4}{5}$.) Two electrocardiograms (lead *II*) from the same patient as Figs. 145 and 146, showing the onset of *A-V* rhythm, its continuation and the first auricular response to the returning *S-A* rhythm. During the phase of established *A-V* rhythm, the auricular complex *P* is buried in the corresponding ventricular systole and is not visible. Time in thirtieths of a second.

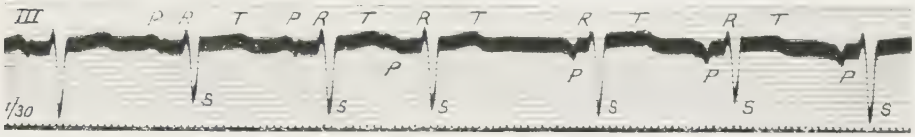


Fig. 149. ($\times \frac{4}{5}$.) An electrocardiogram from lead *III*, showing three responses of the heart to the *S-A* rhythm, an extrasystole, and the first three responses of an established *A-V* rhythm. When the *A-V* rhythm appears, the *P-R* interval shortens and *P* becomes inverted. In this example the *A-V* rhythm is from a high level of the node, higher than in the case in Fig. 148, and the systoles of the auricle and ventricle do not begin simultaneously; the reduction of the *P-R* interval is slight. Time in thirtieths of a second.

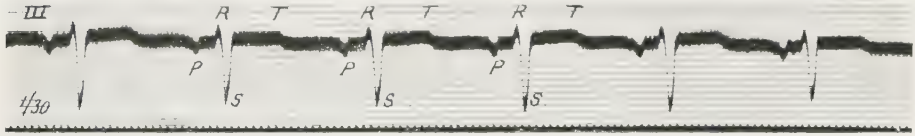


Fig. 150. ($\times \frac{4}{5}$.) This figure is from the same patient, and displays the same *A-V* rhythm as does Fig. 149. Time in thirtieths of a second.

most examples there is a waxing and a waning in the rate of the *S-A* rhythm, and as the heart slows the *S-A* node loses control and the *A-V* node captures it; while with its quickening the *S-A* rhythm once more asserts itself (Fig. 145 and 146).

Interesting examples of *A-V* rhythm occurring during the vagal slowing accompanying expiration have been published by Wilson (*781*) and others (*193*). The former writer has shown also that normal subjects often develop *A-V* rhythm as they are passing under the influence of atropine, the new rhythm appearing spontaneously or under vagal pressure. He suggests that in such cases the vagal endings in the *A-V* node are released first by a selective action of the drug. Escape of the node is not uncommon as a

result of simple vagal stimulation in man. It seems clear that such temporary dislocations of the pacemaker are due to reduced activity of the *S-A* node; but all instances are not of this kind, it would seem, for full atropinisation will not always re-establish the control of the *S-A* node (*193*). In these the new rhythm is probably due to heightened activity of the *A-V* node.

Clinical electrocardiograms of *A-V* rhythm are shown in Fig. 147 and 148. Fig. 147 shows the onset of *A-V* rhythm; the auricular responses to the *S-A* rhythm are undisturbed, but the last two ventricular beats of the curve are responses to the *A-V* node. Later (Fig. 148) simultaneous contraction of auricle and ventricle becomes established; this is not clear in the electrocardiogram, for the auricular complex is hidden by the ventricular. At the end of the same curve the *S-A* rhythm is appearing once again. Another example of clinical *A-V* rhythm, taken from a patient who displayed it almost constantly, is seen in Fig. 149 and 150. In the former of these curves the *S-A* rhythm is shown for three cycles; an extrastole of auricular origin interrupts the regular action of the heart and this disturbance is followed by a new rhythm in which the *P-R* interval is shortened and *P* is inverted in the electrocardiogram. A similar rhythm is shown in Fig. 150, which is representative of the curves usually taken from this patient. In this case the *A-V* rhythm comes from a high level of the node. Admirable examples of *A-V* rhythm in which ventricular contraction preceded the auricular have recently been published by Mathewson (*544*).

Fig. 151. (*Heart, 1914, V, 247, Fig. 5.*) A clinical curve, for comparison with Fig. 141. The *S-A* rhythm is slowing and the *A-V* node gains control. The transition in the shape of auricular complexes is a gradual one; this is due to the response of the auricular muscle to both centres for two or three cycles. Time in fifths of a second.

Curious transitions from *S-A* to *A-V* rhythm in patients are sometimes recorded electrocardiographically. An example is shown in Fig. 151, and it should be compared with Fig. 141, page 187. Three cycles of *S-A* rhythm are shown at the beginning of the curve, and a single cycle of *A-V* rhythm (the *P-R* interval is shortened and *P* is inverted) is seen at the end of the curve. Between these are three cycles in each of which the ventricle responds to the *A-V* node, but in which the auricle responds simultaneously to impulses entering it from both nodes. During the first of the three cycles the main mass of the auricle responds to the *S-A* node and *P* is but little

changed, during the next cycle a greater portion of the auricle responds to the *A-V* node and in the third cycle a greater portion still. These events are reflected by the shape of *P* as it is traced from one cycle to the next.

That clinical *A-V* rhythm is under vagal control has been shown by White (*766*). In his case of established *A-V* rhythm the rate responded to exercise, forced respiration, vagal pressure and atropine very readily. The same worker noticed that under vagal pressure the *Vs-As* interval lengthened, which conforms to the experimental findings.

CHAPTER XVI.

NEW RHYTHM CENTRES (IDIO-VENTRICULAR RHYTHM, ETC.).

When the auricles and ventricles are dissociated, as by direct injuries of the *A-V* bundle, the ventricle assumes a rhythm of its own. This has been termed the *idio-ventricular* rhythm. It is dormant when the heart is beating naturally, in certain respects resembling, and in certain respects differing from, the atrio-ventricular rhythm. It may be stated as a general law that if several heart centres are simultaneously active, the heart, as a whole, will be dominated by the centre which develops its rhythmic impulses most rapidly. It is for this reason that the *S-A* node controls the naturally beating heart, for the rate at which impulses are built up in this node is greater than in any other centre in the organ.

If a rhythm, more rapid than the natural, is excited by stimulating a heart, the natural rhythm becomes submerged, to reappear immediately stimulation ceases. This submerging of the natural rhythm is explained by supposing that a contraction wave which passes over the heart discharges any partially developed impulses in the musculature. So, if rapid rhythmic contractions are forced by stimulation, natural impulses developing at a slower rate never reach maturity, and therefore fail to provoke contractions while the artificial rhythm is in progress. Similarly, an *A-V* rhythm is not displayed by a normal heart because it is of a lower order than the *S-A* rhythm ; its rate is less. This fact sufficiently explains the normal subjection of the *A-V* node. The still lower rate at which the idio-ventricular centre produces its rhythm would be sufficient to account for the usual subserviency of this centre both to the *S-A* and *A-V* nodes. Destroy the *S-A* node and the *A-V* node promptly secures control ; but the heart beats at a slower rate. Destroy the junction between *A-V* node and ventricle and the ventricle beats more slowly still and in response to intrinsic impulses (idio-ventricular rhythm).

But in the case of this idio-ventricular rhythm there appears to be an additional factor. The rate of this rhythm while it is developing is influenced by the rate at which the ventricle was beating previously. Sever the *A-V* bundle abruptly and the ventricle remains quiescent for a considerable period—it may be a minute or more—and the new rhythm develops slowly, increasing in rate as it proceeds. It was for this reason that Gaskell (*215*), observing this phenomenon in the frog's heart, termed the rhythm a

"rhythm of development." Erlanger (*150*) noted that if the bundle is compressed gradually, thereby reducing the number of ventricular responses gradually, the idio-ventricular rhythm develops its full rate more speedily. The same feature was displayed by these workers in another fashion. If the rhythm has fully developed, and the ventricle is stimulated and made to beat rapidly, then, at the cessation of stimulation, the ventricle becomes quiescent and the development of the returning rhythm is gradual. But the length of the pause, and the subsequent rate of quickening, is controlled by the rate of preceding stimulation and by its duration; the centre being less active after rapid or long stimulation of the muscle. Cushny (*90*), who has confirmed these facts, explains them by supposing that the new centre is fatigued by the receipt of extraneous contraction waves. A similar fatigue is not observed in the case of the *S-A* node or in the case of the *A-V* node providing these centres are well nourished.*

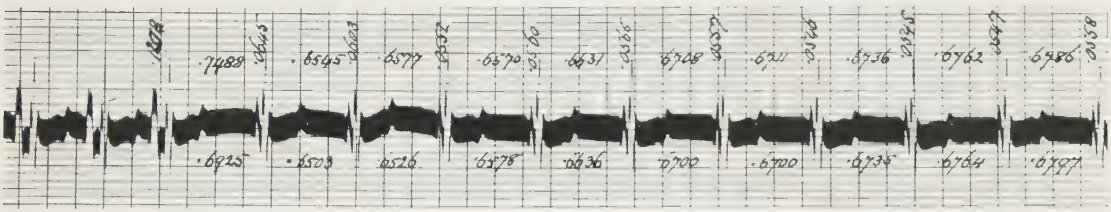


Fig. 152. (*Heart, 1913-14, V, 335, Fig. 16.*) An electrocardiogram from a dog in which the sulcus was cooled and an *A-V* rhythm developed. The auricle was stimulated by successive induction shocks at a rate sufficiently rapid to submerge the *A-V* rhythm. At the cessation of stimulation the *A-V* rhythm at once took control (4th cycle of the curve), but the rate of the *A-V* rhythm declined until it reached the rate shown before stimulation. The figures on the curve are the intervals in decimal points of a second; inter-auricular intervals above, inter-ventricular intervals below, and *As-Vs* intervals written vertically. Time in fifths of a second.

Origin of the idio-ventricular rhythm.

The new rhythm which controls the movements of the dissociated ventricle appears to have its origin in the uninjured portion of the bundle directly below the seat of injury. That the bundle below the injury continues its function is suggested by the maintenance of its structure; it does not degenerate (*374*), an observation which has been made both

* On the other hand, the *S-A* node and the *A-V* node (Fig. 152) often appear to be stimulated rather than fatigued (*490*), and in this respect therefore, there is a contrast between these centres and that responsible for the idio-ventricular rhythm which develops after section of the bundle. Exceptionally, and when there is reason to believe that the heart is poorly nourished and in a hypodynamic condition, the *S-A* node or the *A-V* node may exhibit similar fatigue to that of the idio-ventricular centre (see Fig. 210, page 244).

clinically and experimentally (*148*). If complete heart-block is obtained experimentally by cooling the region of the *A-V* node, and if subsequent to the block being established cooling is continued, standstill of the ventricle is said to occur (*41*). It is to be assumed that in this experiment, the new impulse centre is so near to the region cooled as eventually to be involved in it. Satisfactory evidence for the belief that the new rhythm arises in the bundle is derived from electrocardiography. The natural form of ventricular complex is preserved (see Fig. 114, page 163, and Fig. 135, page 177); the complex is the same before and after the injury, indicating that in the latter circumstance the beat is still of supraventricular origin. It arises between the lesion and the division of the bundle into its two branches, and the excitation wave is distributed to the ventricle along the accustomed channels. If, in addition to the crush of the bundle, a main bundle branch is cut, the resultant ventricular curve is similar in every respect to that obtaining where the second lesion alone has been produced (*475*). Thus the lesion in the division of the bundle interferes with the spread of the excitation wave in precisely the same fashion, whether the wave originates in the auricle, or is derived from the idio-ventricular impulse centre. Clearly, therefore, the latter arises above the bundle division.

The ventricular rhythm in dissociation, produced, not by lesions, but by the action of other agencies (for example, adrenalin, asphyxia, digitalis, anaphylaxis, etc.), is also known, from the form of the electrocardiogram, to have a supraventricular origin. But in these instances, it is not so clear that the bundle is responsible. Thus in the complete heart-block of asphyxia, a higher origin in the *A-V* node has been determined. If a cat is asphyxiated while *A-V* rhythm is in progress, the reversed form of heart-block appears; the auricle, and not the ventricle, fails to respond. When the block becomes complete, the auricle no longer beats, but the ventricular rhythm continues undisturbed *and at its previous rate* (*482*). From this we may conclude that the centre forming the ventricular impulses remains unchanged and is situated, as at the beginning of the experiment, in the *A-V* node. The region of block in asphyxia is above this rhythm centre.

To sum up, it may be said that the rhythm governing the ventricle probably arises immediately below that region of the junctional tissues upon which the injury falls, be the injury physical, chemical or nervous or be it situate in the upper or lower reaches of the tract.

The influence of the vagi upon idio-ventricular rhythm is considered in Chapter XXXI.

Escape of new centres; ectopic impulses.

It is convenient to describe the response of the heart, in part or in whole, to impulses discharged by a new centre as an "escape" of that centre, when the rhythm of the new centre is of the same genetic type as the physiological rhythm of the *S-A* node. Such escape is conditioned if the

rate of impulse formation in the new centre exceeds the rate at which impulses are received in that centre from outside. Usually it is due to a change in the relative activities of the old centre and the new, that of the old centre being depressed or that of the new centre being exalted (*284*); and of these alternatives, the former is much more common than the latter.* Escape may happen also (as in *A-V* dissociation) when the path of conduction from the old centre to new is interrupted.

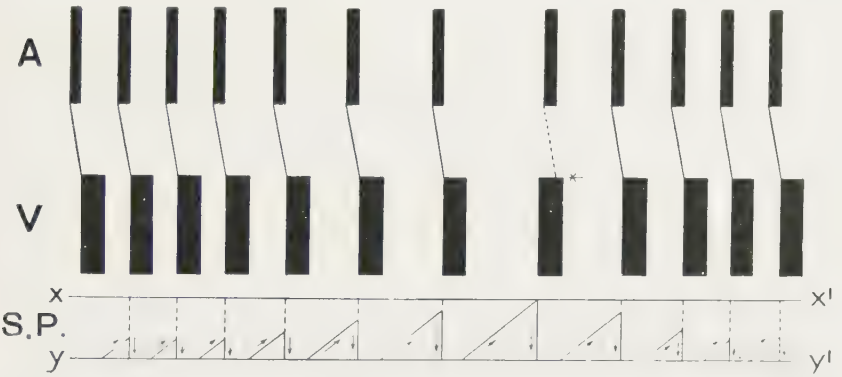


Fig. 153. A diagram illustrating ventricular escape, when the sinus rhythm is sufficiently retarded and when the conditions predispose to such escape. It is supposed that stimulus production (*S.P.*) occurs at a constant rate in the ventricular centre, but that as a rule the pauses are of insufficient duration to allow the stimulus material to accumulate to the critical point (the line *x*, *x'*) at which the growing impulses are discharged. At one point in the figure a spontaneous ventricular beat (marked with an asterisk) is represented, where the pause is longest.

Fig. 154. ($\times \frac{2}{3}$.) A polygraph curve illustrating single escape of the *A.V.* node in a patient who exhibited persistently slow heart rate. At the escape, auricle and ventricle contract together. The escape occurs at the end of the longest pulse beat. It is known to be an escape of this node because the tracing is from the same case as are Fig. 145 and 146.

The simpler forms of escape are those in which the old heart rhythm is submerged by a rhythm emanating from a new centre. The examples cited so far have been of this kind. But escape may be a much more transient event; there may be escape for a single heart cycle only. The

* Examples of ventricular escape in which the rate of the escaping ventricle rises above that of the auricle are rare; in such cases the ventricle and auricle beat for the most part independently, but the rate of the ventricle is the faster (see White and Heard and Colwell) (*244*, *767*). This disorder has been seen in patients under the influence of digitalis.

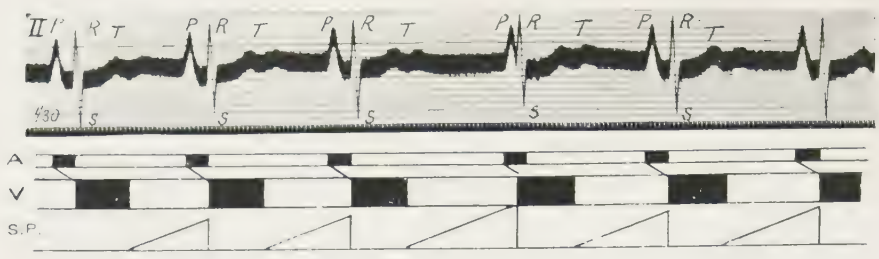


Fig. 155. ($\times \frac{9}{10}$.) A single escape of the ventricle, conditioned by slowing of the *S-A* rhythm and shown electrocardiographically. Each beat of the auricle is awakened by natural (*S-A*) impulses. The fourth ventricular contraction is not in response to the auricle but to a new centre. The diagram below the curve shows the time-relations and origins of the auricular and ventricular systoles. The zig-zag line (*S-P*) represents the building-up of the impulses which from time to time are responsible for ventricular escape.

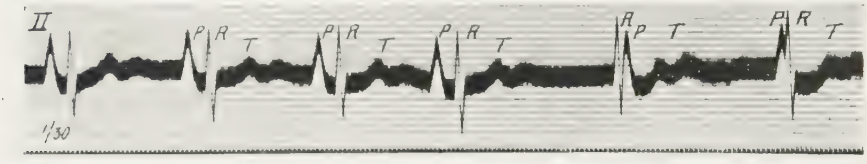


Fig. 156. ($\times \frac{9}{10}$.) A similar curve from the same patient, showing repeated escape of the ventricle. Time in both curves in thirtieths of a second.

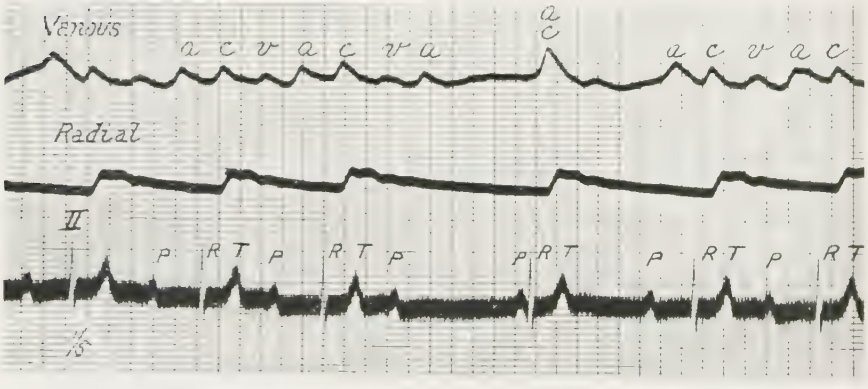


Fig. 157. ($\times \frac{5}{6}$.) Venous, radial and electrocardiographic curves taken simultaneously from a patient and showing heart-block. The *As-Vs* interval is prolonged and at one point the ventricle fails to respond, a long pause ensues and during this pause the ventricle escapes. In this cycle auricular and ventricular systoles fall partly together ; the corresponding *P-R* interval is very short and an exaggerated wave $\frac{a}{c}$ appears in the neck. Time in fifths of a second.

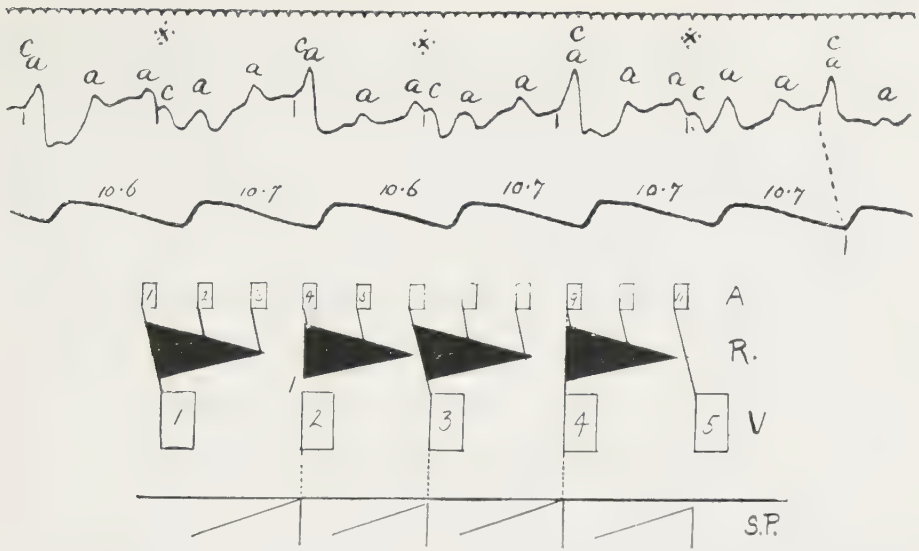


Fig. 158. (*Quart. Journ. Med., 1908-09, II, 361, Fig. 8 and 9.*) A polygraph curve illustrating escape of the ventricle at alternate beats. The ventricle escapes whenever the opportunity is afforded to it. Partial heart-block is present and the ventricle is wavering between a 2 : 1 and 3 : 1 response. The black triangles represent impaired conduction in the bundle, the depth of the triangle at any point represents the measure of this impairment. After the first response of the ventricle, conduction is so far impaired that two auricular impulses (A^2 and A^3) fail to reach the ventricle; the next (A^4) would produce a response but it is anticipated by the ventricular centre. This slightly premature contraction of the ventricle enables the bundle to recover its conductivity and to pass the sixth auricular impulse (A^6) to the ventricle. The same events are then repeated. The result is an almost regular ventricular action, alternate cycles being a trifle short; these are terminated by responses to the auricle; the alternate and longer cycles are terminated by escaped beats of the ventricle. This is an example of escape of the idio-ventricular centre, for curves from the same patient frequently showed the usual picture of dissociation.

centre which escapes is either the *A-V* node or the bundle below it, for the escaped beats are of supraventricular type. In the case of isolated escape a nearer localisation is not always possible, but it is clear that many instances hitherto regarded as "ventricular escape"—a term hitherto implying the activity of the idio-ventricular centre—are in reality due to *A-V* nodal escape (see Fig. 154). I use the term "ventricular escape" in the present chapter in a more general sense, and without wishing to infer that the impulse necessarily arises in the ventricle. The mechanism may be illustrated diagrammatically. The auricular and ventricular contractions are indicated by the rectangles *A* and *V* respectively (Fig. 153). Stimulus production in a new centre is represented by the rising and falling line *S.P.* It is supposed that the auricle is beating in response to impulses produced in the *S-A* node. At the same time the new centre is elaborating impulses at a constant rate. At the beginning and ending of the tracing, when the *S-A* rhythm is rapid, the ventricle has no chance to escape,

the impulses being discharged while they are still immature. In the middle of the figure, where the *S-A* rhythm slows sufficiently, a single impulse develops fully and, discharging, creates a ventricular response which anticipates the response of the ventricle to the oncoming auricular impulse. Solitary escape of this kind is frequent clinically (*354*, *427*, *480*, *631*). It was first described by Mackenzie (*502*), and a notable example has been recorded by Wenckebach (*762*).

Illustrative examples are seen in Fig. 154 to 157. It is to be noted that such solitary escape of the ventricle comes when the ventricle is starved of its natural impulses to contract, either by slowing of the *S-A* rhythm (Fig. 155) or by *A-V* heart-block (Fig. 157); and in a given patient it is often a feature that the escaped contractions terminate all diastoles *of more than a given length* and terminate these only (*414*, *427*, *480*, *762*). This is explained if we assume that the impulses which occasionally excite the ventricle are built up persistently and at a constant rate (Fig. 158); whenever a sufficient time interval elapses an impulse matures and, discharging, excites the ventricle.

Escape of the ventricle accounts in part for the rarity of high grades of partial heart-block; 2 : 1 block is common; 3 : 1 block is rare.* Curious disorders of conduction in which 2 : 1 and 3 : 1 cycles are mixed with spontaneous ventricular beats are on record (*480*); and in instances where there are successive escapes of the ventricle the picture which Erlanger terms "relative complete heart-block" is produced. These instances are certainly instances of idio-ventricular escape; they pass insensibly into dissociation. Instances are on record also of alternate responses of the ventricle to auricular and to new impulses in which the pulse may be almost if not quite regular. An example of this kind is shown in Fig. 158. A not dissimilar instance, associated with factitious alternation of the pulse, has recently been recorded (*713*).

The reason for escape of a new centre is not always clear, or rather the reason for the change in the relative activities of the centres, the one emerging, the other submerging, is not always easy to define. Thus, if a given heart rhythm is interrupted by a single premature beat or by a number of such beats following each other successively, another rhythm centre may be stimulated to an unusual, though transient, wakefulness (*490*).

In Fig. 159 a rhythm of *A-V* nodal origin is interrupted by a beat forced from the ventricle; after the disturbance, the *S-A* rhythm predominates for a short while, its rate being temporarily enhanced through unknown channels. A clinical example of escape of the *A-V* node, consequent upon a premature contraction originating in the auricle, is to be found in Fig. 149, page 193. Another clinical instance of escape, though of a slightly different kind, is to be seen in Fig. 160. In this patient (*451*), premature contractions, arising in an abnormal auricular focus, were frequent, and it

* There is another cause for the rarity of 3 : 1 block, though we are not aware of its nature. In experiment 4 : 1 block is much more frequent than 3 : 1 block.

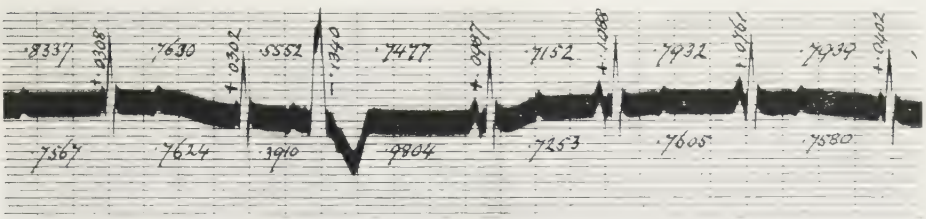


Fig. 159. (*Heart, 1913-14*, v, *335*, *Fig. 9.*) An electrocardiogram from a dog. *A-V* rhythm is interrupted by a beat forced from the right ventricle. An escape of the *S-A* node succeeds this disturbance and is associated with accelerated action of the heart. As the heart rate slows again, *A-V* rhythm is resumed. Time in fifths of a second.

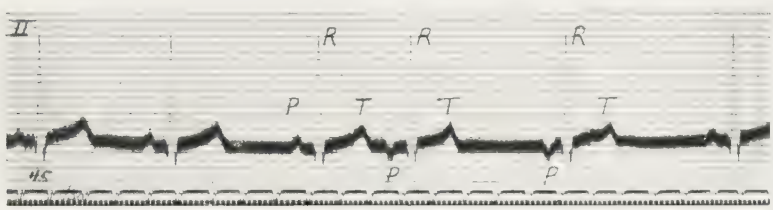


Fig. 160. A single premature contraction of auricular origin in a patient. The first beat after the pause originates at an abnormal auricular focus, as does the premature contraction. Time in thirtieths and fifths of a second.

nearly always happened, as in the present instance, that the first beat following the disturbance was generated from the same or a neighbouring and unnatural focus.

Centres of rhythm production.

The natural pacemaker, the *S-A* node, is endowed with higher rhythmicity than any other portion of the cardiac musculature. Of other centres capable of usurping this function in pathological conditions there are several. Of these *ectopic** centres, the *A-V* node, of similar structure, stands first. There are many reasons for believing that all intact portions of either of these it nodes are possessed of rhythmic activity. In destructive experiments has been shown necessary to eradicate the whole *S-A* node to ensure the inactivity of this region of the auricle (*71*) ; in my own experience, I have found cooling of the head of the node insufficient to induce *A-V* rhythm in dogs, which develops whenever the temperature of the node is reduced in its whole

* Hering uses the term *heterotopic* in the same sense. By the adjective ectopic I wish to express in this and subsequent chapters the origin of beats, whatever their nature, from a focus other than the *S-A* node, and to convey no more than that in respect of their qualities.

length ; stimulation of the vagus often depresses rhythmicity in the head of the *S-A* node and permits the escape of slower rhythmic impulses from its tail (*483*).

It is probable that the same universality of rhythmic function is possessed by various parts of the *A-V* node ; many suggestive reasons for this belief have been discussed.

It is not proved that any parts of the auricular muscle other than those mentioned have enough inherent rhythmic power to control and maintain the heart beat. Erlanger's experiments (*145*, *147*), it is true, profess to show that many regions of the right chamber and septum may produce rhythmic impulses under given conditions ; but it is not always clear that portions of one or other node were not included unintentionally in the areas tested.* The left chamber is considered by these writers to be wanting in rhythmicity, though Fredericq (*186*) appears to hold that exceptionally it may be rhythmic. It is certain that if rhythmic power is present in the portions of the mammalian auricle which do not contain nodal tissue, the power is but poorly developed ; and it is probable, even if rhythms may so arise, that they are incapable of maintaining the heart beat for any considerable length of time under natural conditions.

In the ventricle, the bundle is certainly endowed with rhythmic power ; its continuation in branches of similar elemental constitution suggests that these possess similar properties. It is said that the main divisions are readily excited by heat (*205*), and we know that when both divisions of the bundle are cut the ventricle continues to respond to impulses generated within itself. Automaticity in the arborisation has received little or no study ; if it be present universally it can only be so in relatively low degree. The trend of conclusion, as it concerns the complete mammalian heart, is to proclaim the special structures as the chief agents of rhythm ; quite possibly they are the sole agents. Those special structures which are composed of minute muscular elements are particularly, but not exclusively, endowed with the rhythmic function.

* Moorhouse (*562*), writing more recently, states that strips are equally rhythmic whether they contain nodal tissue or not. If that is so, it is difficult to understand why the auricle *in situ* ceases to beat when *S-A* and *A-V* nodes are thrown out of action (*482*, *689*). Hering (*284*) stated that after isolating the two nodes from the mass of right-auricular tissue, the latter continued to beat, and, on the strength of these experiments, concluded quite positively that there are other auricular centres capable of rhythmic action. The hearts, subsequently examined by Koch (*388*), showed the weakness of this conclusion, for nodal tissue was found attached to the mass of auricular muscle. The discussion over these hearts has been continued by Hering (*295*), who still maintains that in one instance his conclusion has obtained justification ; it serves to emphasise the importance of strict histological controls in all experiments of this type.

CHAPTER XVII.

VENTRICULAR EXTRASYSTOLES.

WHEN the ventricle of an animal is stimulated in diastole by a mechanical or electric shock, it responds to the shock and contracts. This power of response to artificial stimuli has been termed *excitability*.* The ventricle is excitable, as Marey (*537*) showed, during the whole of its diastole, but is inexcitable or refractory during the period of its systole. The strength of a response of a ventricle to stimulation obeys the law stated by Bowditch (*38*); it is independent of the strength of stimulation. But the strength of the beat is controlled by the rest which the muscle has enjoyed before a fresh contraction is forced. If the ventricle is excited to contract during its natural diastole by a single induction shock, a disturbance is produced which may be illustrated diagrammatically (Fig. 161). For the first two cycles

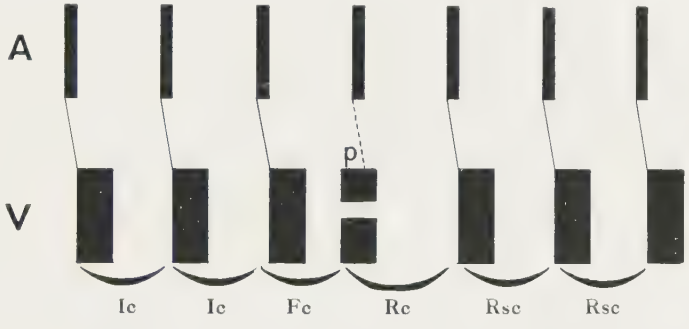


Fig. 161. A diagram illustrating the disturbances of the heart's mechanism when a systole is forced by exciting the ventricle during diastole. *Ic* = initial cycle; *Fc* = forced or extrasystolic cycle; *Rc* = returning cycle; and *Rsc* = restored cycles. *P* is the premature or forced beat. The auricular rhythm remains undisturbed. The forced and returning cycles are together equal in length to two initial cycles.

the heart is represented as beating naturally. During the next ventricular diastole a premature beat *P* is forced by stimulation of the ventricle. This forced beat is followed by a diastole of unusual length and then the normal heart beats return. It will be convenient for descriptive purposes to name the several cycles of this figure; calling the first cycles "initial cycles" (*Ic*); the short cycle, the "extrasystolic cycle" or "forced cycle" (*Fc*), according as the beat which ends it is

* The term is also used very generally, but I think inadvisedly, to cover the power of response to natural impulses. Of the last we have no measure, consequently I use the term in its more restricted sense.

spontaneous or forced by stimulation; the long cycle, the "returning cycle" (*Rc*), and the remainder the "restored cycles" (*Rsc*). The systoles of the figure may be conveniently qualified by the adjectives applied to cycles which precede them. It was first pointed out by Knoll (*381*), that the forced and returning cycles are together equal to two initial cycles; the explanation of this fact we owe to Engelmann (*128*), who worked with the frog's heart. It is that the premature or forced beat has arisen independently

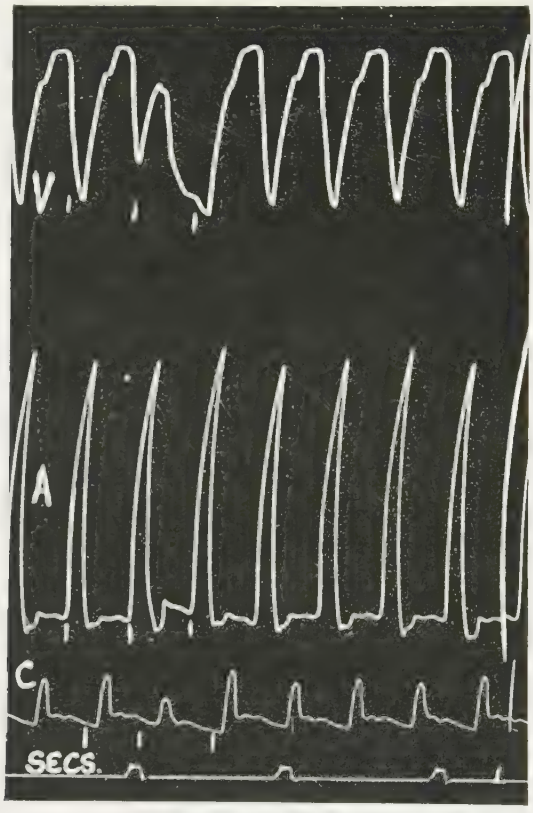


Fig. 162 Myocardiographic curves (*V* = ventricle, *A* = auricle) and Hürthle carotid pressure curve (*C*), from a dog in which the right coronary artery had been tied. Showing a single spontaneous and premature contraction of the ventricle. The pause following the weak beat in the arterial curve is compensatory; the auricular beats maintain their rhythm. The prematurity of the carotid beat is barely perceptible on account of the temporary increase of presphygmic interval. Time in seconds.

of an auricular contraction, and that the succeeding auricular impulse (represented in the diagram by the dotted oblique line) falls upon the ventricle when it is in a refractory state (*refractory period*). As a consequence, the ventricle fails to respond, and awaits the call of the next auricular impulse. The cycle which follows the premature contraction is therefore prolonged, and

inasmuch as it makes amends, by its length, for the shortness of the preceding cycle, its diastole is termed the *compensatory pause*. The same phenomena were observed in the mammalian heart by Cushny and Matthews (*96*). An experimental example of a premature ventricular contraction is shown in Fig. 162. In this figure the rhythm of the auricle is undisturbed ; the rhythm of the ventricle is disturbed temporarily. The returning and restored contractions of the ventricle fall where they would fall had there been no disturbance. Such is the rule. But not infrequently they may be a little displaced ; the systoles of the ventricle, which succeed the disturbance, appear a little earlier than is anticipated, when conduction from auricle to ventricle is quickened, as it may be, after the unusual rest of the long pause. This change in the conduction interval (*As-Vs*) is illustrated in an exaggerated form in Fig. 163.

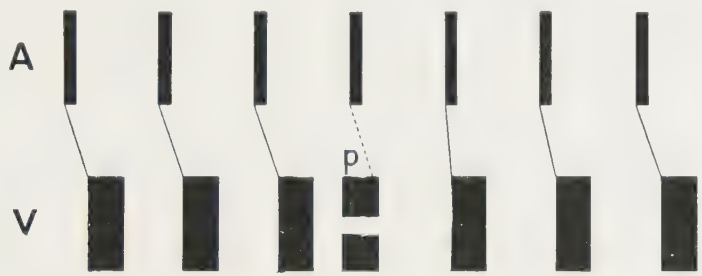


Fig. 163. Similar events to those shown in Fig. 161, but showing some displacement of the beats which follow the forced contraction (*P*) as a result of change of conduction intervals (*As-Vs*). The returning cycle is long, and the pause rests the conducting tissues ; when the heart resumes its harmonious beating the conduction intervals are at first shortened by reason of this rest.

Another variation of the events may be seen from time to time. Of the rhythmic auricular beats, one customarily fails to produce a corresponding beat of the ventricle. It fails because the impulse conveyed from auricle to ventricle falls during the refractory period created by the premature systole. But if the heart's action is slow and the forced beat occurs at an early period of diastole, the forced systole may terminate before the rhythmic impulse from the auricle becomes due (*584*) (see diagram below, Fig. 164).

In such a case the ventricle responds to each auricular impulse, and also contracts in response to the unusual excitation. This form of disturbance is termed an *interpolated* extrasystole (*104, 411, 704*).* The *As-Vs* interval

* The reason why many of these extrasystoles fail to propagate themselves to the auricle, producing a retrograde beat, remains mysterious. In some instances the retrograde impulse is calculated to coincide with the next natural auricular impulse, but this is not always the case.

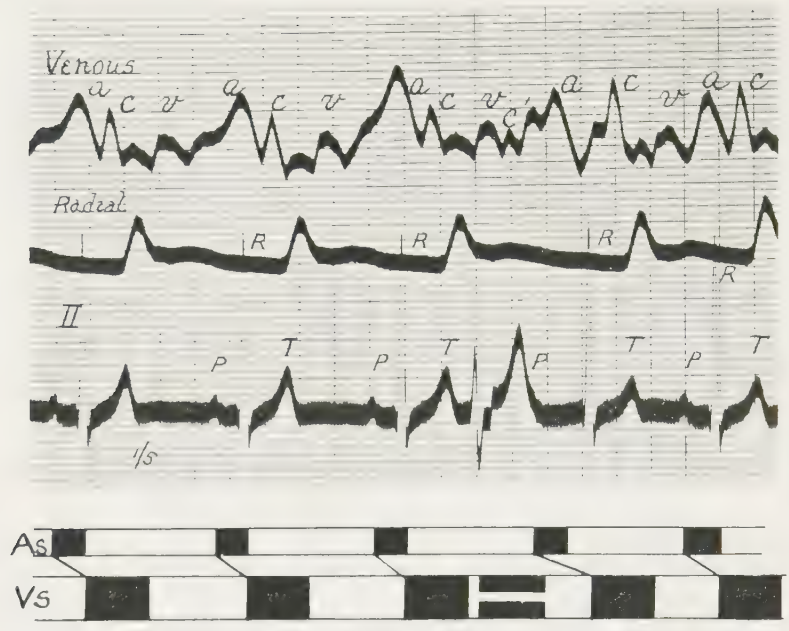


Fig. 164. Venous, radial and electrocardiographic curves from a patient, exhibiting an *interpolated* extrasystole of the ventricle. The first two cycles are natural; the diastole of the third is disturbed by a premature beat of the ventricle; the succeeding auricular impulse yields a ventricular response, but, as the rest has been short, the corresponding *As-Vs* interval is lengthened somewhat. The *P* summit of the returning cycle is obscured because it falls on the end of the anomalous ventricular complex. Time in fifths of a second.

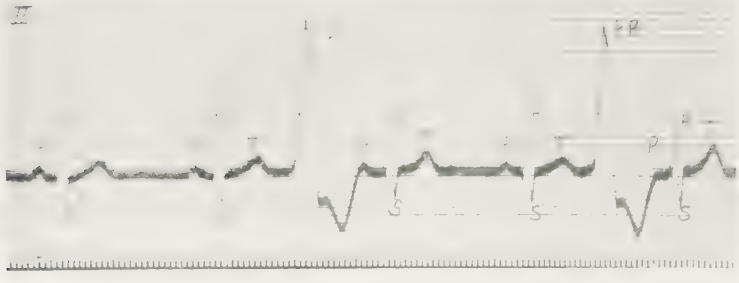


Fig. 165. Human curve. Premature contractions arising in the right or basal portions of the ventricle and interpolated between normal heart cycles. Time in thirtieths of a second.

following the interpolation is usually, though it is not always, materially increased.*

The disturbances of the heart's mechanism which are seen when the ventricle is stimulated artificially are faithfully reproduced when, in man, premature contractions of the ventricle come spontaneously and when, in animals, they appear in response to the introduction of toxic substances into the circulation. Because there is this likeness in detail, spontaneous extrasystoles are often ascribed to an unusually excitable condition of the ventricle. This conclusion or inference is not justified ; it is not proved that extrasystoles as they occur spontaneously are due to disturbed excitability of the heart (see Chapter XXIX).

Records of ventricular extrasystoles.

It was Cushny (*86*) and Wenckebach (*754*, *755*) who first suggested that a certain form of pulse intermission seen in the human subject is similar, in so far as the disturbed order of chamber sequence is concerned,to the intermission of the pulse produced experimentally by stimulating the ventricle. The proof that the two disturbances are alike came when Mackenzie published his early venous curves (*500*, *501*).

Arterial records.—The rhythm of the pulse is disturbed by an intermission. In most instances, this long pulse cycle has exactly twice the duration of the usual pulse cycle. The pulse shows, on measurement, that the dominant rhythm is undisturbed.

The long pulse pause may or may not show a trace of the premature ventricular contraction. The premature beat of the ventricle is weak since the contractile power of the heart has had insufficient rest fully to recuperate. The weak expression of the contraction in the arteriogram is in part the outcome of this unrestored power ; it is in part due to relative emptiness of the ventricle when the latter is called upon to contract. If the premature beat occurs sufficiently early, it may be so weak, or the blood content of the ventricle may be so small, that the aortic valves are not raised ; in that case a second heart sound is not produced and the pulse is not affected by the extrasystole (Fig. 166). Occurring later in diastole the extrasystole affects the pulse and, according to the power of the contraction and the amount of blood evacuated, the extrasystole expresses itself as a minute wave or as an almost fully developed pulse (Fig. 167). The returning contraction of the ventricle, *i.e.*, that which follows the long pause, is powerful and the pulse prominent, for reasons the reverse of those which have been considered (*626*). Premature beats are always accompanied by a movement of the heart's apex and by a first heart sound, though the latter is often modified.

* The reason of this prolongation is also obscure seeing that the extra beat has not been propagated through the *A-V* bundle. A premature beat, forced from the ventricle in experiment during partial *A-V* block, often heightens the degree of block notably though the beat is not propagated to the auricle (*484*, *570*).

The presphygmic interval of the weak beat is usually longer than those of normal beats in the same case, and so the degree of prematurity is not always fully displayed in the pulse (see Fig. 162).

Interpolated extrasystoles usually fail to affect the arterial pulse (see Fig. 164); when an arterial pulsation does occur, the next pulse wave is diminished in size. The larger the pulse of the interpolated beat, the smaller is the succeeding pulse. When, as sometimes happens, the interpolated arterial pulse is equal to the succeeding pulse beat, the two have the appearance of a pair of extrasystoles, from which oftentimes they may only be distinguished by electrocardiography.

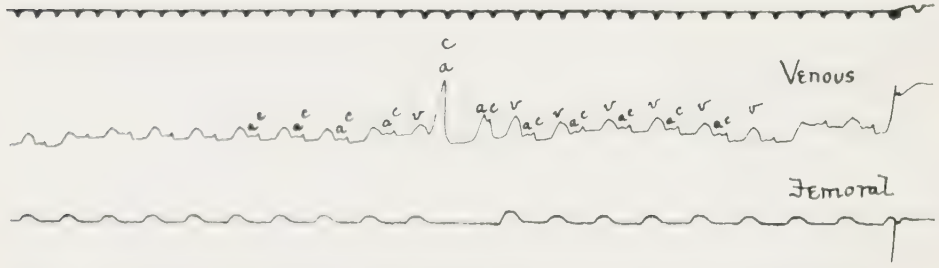


Fig. 166. Venous and femoral curves from a dog, showing the effects of a single premature ventricular contraction induced by electrical stimulation of the ventricle. There is no sign of the early beat in the arterial curve; it falls at the same time as the anticipated *a* wave and gives rise to an exaggerated wave $\frac{a}{v}$ in the phlebogram.

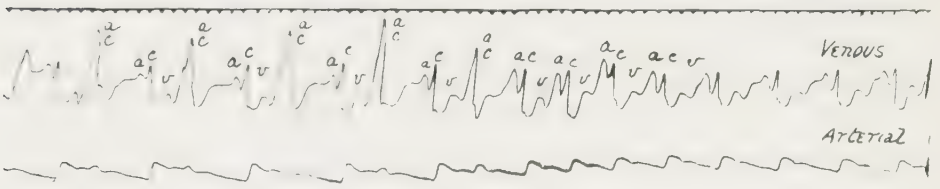


Fig. 167. ($\times \frac{2}{3}$.) Extrasystoles of ventricular origin in a patient. The last part of the tracing displays a normal heart action. The first part shows extrasystoles, alternating with normal cycles. A rhythmic auricular beat falls with each extrasystole and produces an exaggerated wave $\frac{a}{c}$ in the venous curve.

Venous curves.—These show the undisturbed sequence of the auricular contractions (Fig. 166 and 167). Where the premature ventricular beat falls synchronously with an auricular systole (see Fig. 175), an exaggerated wave $\frac{a}{c}$ is produced.

Electrocardiograms.—When a premature beat arises spontaneously in the ventricle or is forced in an experiment, the electrocardiogram usually displays every contraction of auricle and of ventricle, and the full analysis of chamber contraction may be read in these curves alone. The auricular complexes

are of constant form throughout (Fig. 168); the ventricular complex of the forced beat is anomalous and varies in form according to the area of muscle in which it has arisen. In these electric curves the anomalous ventricular complex falls simultaneously with the rhythmic auricular complex, and the two are superimposed (see page 160, Fig. 110 and 111, and explanations).

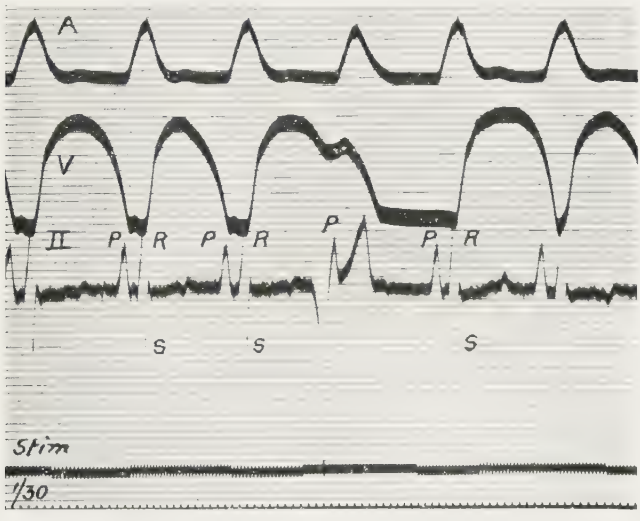


Fig. 168. Myocardiographic curves (*A*) from the auricle and (*V*) from the ventricle of a dog, accompanied by an electrocardiogram (lead *II*). A single beat has been forced prematurely by stimulation of the apex of the left ventricle and has yielded an anomalous complex. The rhythmic auricular complex falls with this and is superimposed upon it. Time in thirtieths of a second.

Ventricular curves of beats arising from the ventricle.

Forced beats.—In curves taken from an axial lead (such as lead *II*), stimulation of the apex of the dog's left ventricle produces a diphasic curve, of which the first phase is downward and the second phase upward (Type *L*, page 154, Fig. 103). On the other hand, upon stimulating the basal region of the right ventricle, the phases are reversed (Type *R*, page 154, Fig. 104).

Kraus and Nicolai (*390*, *391*), who first recognised these distinct types, regarded them as expressing contractions of the corresponding ventricle, and contractions more or less completely confined to them (*hemisystole*). Their hypothesis as stated in its original form is untenable and, indeed, has been modified by the same writers (*392*). It is true that stimulation of the greater part of the ventral surface of the right ventricle yields a curve of the second type (Type *R*) and of the ventral surface of the left ventricle a curve

of the first type (Type *L*). But, as subsequent writers* have pointed out, the question of type in relation to region stimulated is not so simple as Kraus and Nicolai imagined (*361*, *362*, *475*, *670*, *673*).

The chief facts may be summarised. No two points of stimulation yield precisely the same resultant curve (Fig. 170), and an infinite variety of forms may be obtained from one and the same heart ; but if two points stimulated lie close together, the corresponding curves resemble each other, and the resemblance is the greater the nearer the points of stimulation approach each other. Stimulation of a given point always yields the same ventricular curve, and this is so whether the stimulus falls in early or late diastole (*447*). Now when the ventricle is directly stimulated, the spread of the excitation wave must clearly be abnormal, and to this abnormality of spread, the dissimilarity of the complexes of natural and excited beats is due. Clearly the distribution in the case of two points stimulated will not be very different, providing these points are close to one another. The excitation wave spreads from the point stimulated and travels in every direction radially

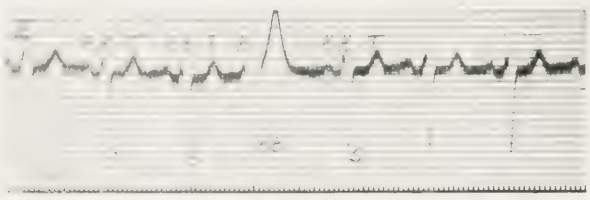


Fig. 169. Premature contraction arising in the left ventricle. A human curve for comparison with Fig. 168.

through the muscle ; it also pierces the thickness of the wall and reaches the Purkinje substance ; reaching this substance, it is propagated with great rapidity over the whole lining of the corresponding chamber, and ultimately spreads to the opposite ventricle.† Thus the ventricle being stimulated, the wave is at first confined to the adjacent muscle, and, until the muscle area involved is considerable, the electrocardiograph, arranged at its usual sensitivity, shows little or no movement. But later the spread is controlled by Purkinje paths, the remainder of the muscle being stimulated through these, and the excitation wave then travels simultaneously from within outwards over a large part of the wall (*475*). This event is signalled by the first conspicuous movement of the string. The ultimate direction of spread therefore is similar to that in the corresponding ventricle when this is activated through normal channels. To this fact, in large part, is due the

* A full summary of the observations up to 1914 will be found in Kahn's monograph (*367*).

† Hemisystole, or a limitation of contraction to one ventricle, has not been shown to occur either experimentally or clinically, see page 183

close resemblance between the curves corresponding to defects in the divisions of the bundle (dextrocardiogram or levocardiogram) and those obtained by stimulating the opposite ventricle (right or left, respectively). The influence of spread and the forms of curves obtained may be illustrated by the details of the following experiment.

The ventral surface of a dog's heart having been exposed (Fig. 170), the muscle was excited at a number of points (1-14), using at each point successive threshold stimuli. The corresponding responses of the heart were recorded, using lead *II*, and single ventricular cycles are shown to the left in the figure. The signal of stimulation was also recorded (see curve 13), and in each instance the time interval between the signal of stimulation and the first prominent deflection was measured. These intervals are tabulated. Subsequently the heart was hardened in a natural condition of diastole and an oblique section was cut passing through the points of stimulation along the line 3-13. A diagram of this section is also represented in the same figure. The distance of the ventricular surface to the Purkinje network was measured in millimetres at all stimulated points. If the series of curves is examined

Point stimulated.	Distance to Purkinje system.	Signal to 1st prominent deflection.
	mm.	sec.
1	R. 4	0·0363
2	R. 3	0·0416
3	R. 3	0·0424
4	R. 4	0·0405
5	R. 3	0·0407
6	R. 3·5	0·0473
7	R. 1·5	0·0293
8	R. 2	0·0245
9	R. 2	0·0343
10	R. 3	0·0497
11	R. 7 L. 8	0·0686
12	R. 12 L. 10	0·0920
13	L. 9·5	0·0920
14	L. 8	0·0699

it will be noticed that the ventricular responses obtained over the whole ventral surface of the right ventricle yield very similar outlines in the electrocardiogram ; these differ from each other in detail and magnitude.* As the point of stimulation is moved along the *A-V* groove from the right towards the left margin and on to the conus (from 1 to 5 in Fig. 170), the excursion of the string, in its response to the heart beat, increases somewhat

* The curves are complicated by the presence of auricular complexes ; these are of the type accompanying natural auricular beats in the case of curves 3, 4, and 5 ; the remaining curves show auricular complexes of the retrograde type.

in magnitude. As the stimulating electrodes are moved from the *A-V* groove (at 3) downwards towards the apex of the heart, the change in form and in magnitude is slight* until the region of the coronary vessel is passed. As the descending branch of the left coronary artery is crossed there is an abrupt change in type; point 11 yields a complex of intermediate type; the complexes from points 12 and 13 have their two chief phases inverted as compared to those obtaining over the right heart. Briefly, the responses from the right ventricle produce electrocardiograms presenting the chief features of dextrocardiograms, while the responses from the left ventricle produce electrocardiograms showing the chief features of levocardiograms. Now there is a relation between the length of the interval (signal of stimulation to first chief phase) and the thickness of the underlying muscle (see Table). In curves 12 and 13, and preceding the chief or downwardly directed movement, there are very evident preliminary phases of a diphasic character. In curves 4, 7, 9 and 10 the complex opens with a steep upstroke, and there is no preliminary phase, but small preliminary phases are distinct in all the remaining curves. These preliminary phases are due to the initial spread in the muscle: for, as they are more or less prominent according as the muscle path is long or short, we may conclude that one of the chief factors governing their appearance is the amount of muscle activated before the Purkinje network begins to convey the impulse. The first chief phase, be it upward or downward, appears immediately after the Purkinje system is involved, and its magnitude is due to quick spread to a relatively large and corresponding muscle area.† Its appearance is delayed according as the muscle layer penetrated is thick. A little consideration will suffice to show that, when the surface of a ventricle is stimulated, only a limited mass of muscle responds to the ingoing wave of excitation; the chief part of the ventricular wall responds to the outgoing wave due to involvement and spread through the Purkinje tissue. The mass responding to the ingoing wave will depend upon the muscle thickness, but it will never be large, because the velocity of conduction in Purkinje tissue is relatively very great. Thus it happens that the direction of travel in the ventricular tissue cannot be controlled by altering the point of stimulation. Stimulation at the base or apex of the right ventricle produces the same end result, an excitation wave travelling from within outwards over the greater part of the ventricular substance. This accounts for the general similarity of curves obtained when stimulating a large area of the right, or when stimulating a large area of the left ventricular surface. The second point to emphasise is the relatively abrupt transition from the type shown in curve 10 to that shown in curve 12. Clearly such transitions are due to

* Sometimes the change is greater than here shown (*666*), the greater or lesser change depending to some extent upon the line along which points of stimulation are chosen.

† In extrasystolic curves, as they are seen clinically, preliminary phases are scarcely ever to be distinguished. Their absence suggests that the extrasystoles arise in the Purkinje tissue and not in the ventricular muscle.

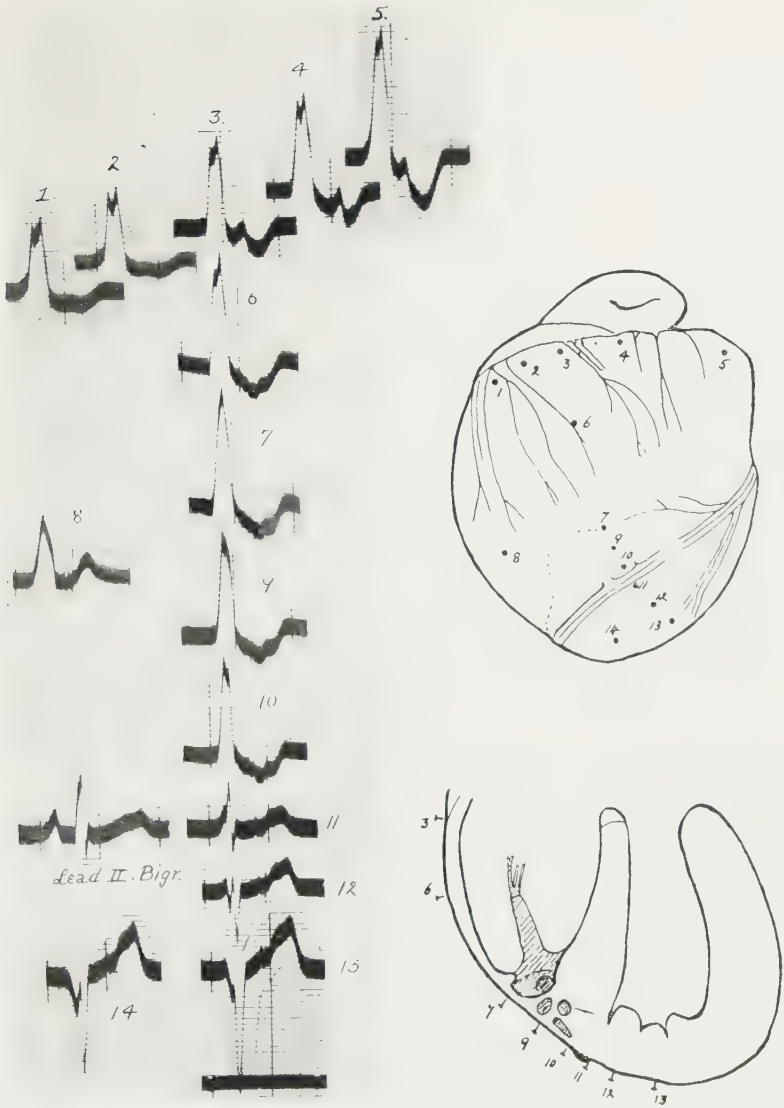


Fig. 170. (*Phil. Trans., roy. Soc., 1916, CCVII, 279, Part IV, Fig. 10.*) A series of curves ($\frac{7}{10}$ nat. size) taken from lead *II* in a dog and resulting from stimuli applied at the corresponding numbered points in the upper outline drawing of the dog's heart. The lower outline is a section of the same heart through points 3 to 13 ($\frac{7}{10}$ nat. size). The vertical lines cutting the electrocardiograms are the fifth second time lines of the original curves.

spread into the right Purkinje system on the one hand and into the left Purkinje system on the other. Finally, when the point stimulated is immediately to the left of the artery (point 11) curves of intermediate type are obtained, and these curves often resemble natural bicardiograms in the same animal very closely, as Rothberger and Winterberg (*666*) and others have pointed out. The explanation is that the two Purkinje systems are involved almost simultaneously. Thus point 11 lay 7 mm. from the Purkinje network of the left ventricle and 8 mm. from that of the right. The electrocardiogram (curve 11) is of dual origin ; it is an algebraic summation of right and left curves and closely resembles the natural bicardiogram in outline.

The type of curve yielded by stimulating the surface of the ventricle seems to depend upon two chief factors, the relation of the point stimulated to the two networks of Purkinje and its relation to the mass of ventricular muscle as a whole. Of these two factors the first exerts the dominant influence.

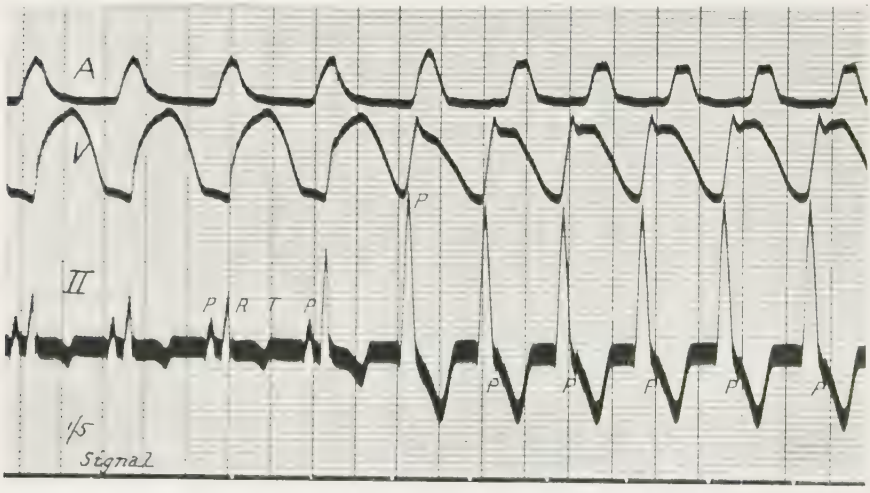


Fig. 171. ($\times\frac{9}{10}$.) Simultaneous curves from a dog's heart. A = auricular and V = ventricular myocardiogram. After three natural cycles the heart responds to rhythmic induction shocks thrown into the right ventricle. The fourth beat of the ventricle in this figure is in part a response to the auricular impulse and in part to the induction shock ; the remaining ventricular beats are pure responses to the rhythmic induction shocks. Time in fifths of a second.

The type of curve naturally changes profoundly with change of lead. We shall understand more clearly the manner in which the excitation wave is distributed in forced contractions when in the future the change of the electrical axis of these contractions has been studied.

Sometimes, when a stimulus enters the ventricle in very late diastole the whole ventricle does not respond to it, some parts of the chamber being

simultaneously excited by the natural impulse descending from the auricle (*451*). In these circumstances the ventricular complex has a transitional outline. Fig. 171 is an experimental curve ; after three natural cycles the heart responds to rhythmic stimuli thrown into the right ventricle (see signal). There are six responses of the ventricle in which the form of curve is fully controlled by the artificial impulse ; these are the last six cycles of the curve. But between these two series stands a ventricular complex of transitional form ; its shape is intermediate between that of the natural complex and the complex corresponding to the excitation wave propagated solely from the point stimulated. The ventricle in this instance has responded partly to the auricular impulse (the *P-R* interval is slightly shortened) and partly to the artificial stimulus. A clinical curve in which a similar interference between two excitation waves is seen in Fig. 171a. The first curve of this kind to be published will be found in my book (see *447, Fig. 120*). In that example a perfect transition from the normal ventricular complex to that fully representing the extrasystolic form of spread is to be seen. A transition due to similar interference, though it is produced in a somewhat different fashion, is to be seen in the clinical example published by Christian (*50, Fig. 10*) ; in this instance the two centres from which the impulses spread were the auricle on the one hand and the ventricle (as an idio-ventricular rhythm) on the other.*

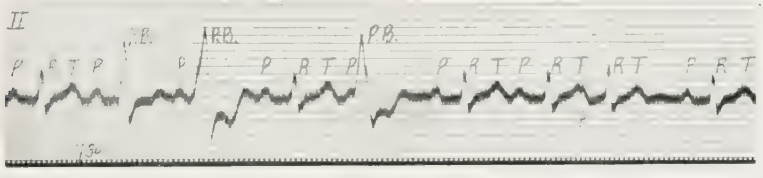


Fig. 171a. (" *Clinical Electrocardiography*," *London, 1913, 1st edit., Fig. 48.*) Premature contractions are shown which arise in the right ventricle late in the ventricular diastole. There are three such contractions (*P-B*) in the curve ; in the second and third instance the excitation wave has spread through the whole ventricle from the point at which the new beats arose. But the first of the abnormal beats has fallen later in diastole, so late in fact that the ventricle responds in part to the natural auricular impulse and in part to the new impulse ; the ventricular complex becomes therefore transitional in type. Time in thirtieths of a second.

The complete length of an anomalous ventricular complex is equal to that of a natural ventricular complex, within small errors of measurement ; this rule holds for all such beats, forced or spontaneous, and is sometimes serviceable in defining the limits of a ventricular complex of odd outline (*447*).

Spontaneous beats.—Clinical examples of ventricular extrasystoles give curves of varied form from case to case, but fall for the most part into two chief categories, the right and the left types (Fig. 172 and 173).

* The events in this curve are quite clear, though they appear not to have been recognised by the writer. The shortening of the intervals between ventricular beats, while the ventricle responds to the auricle alone, should be noticed.

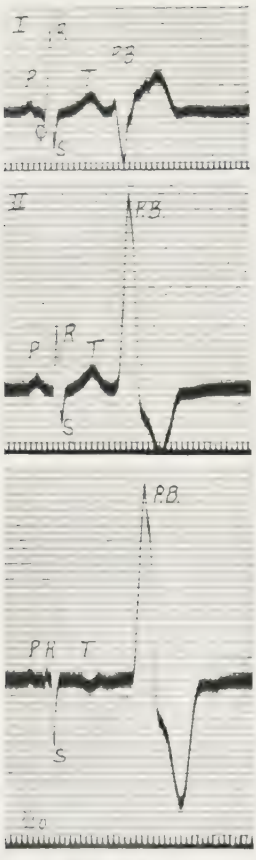


Fig. 172.

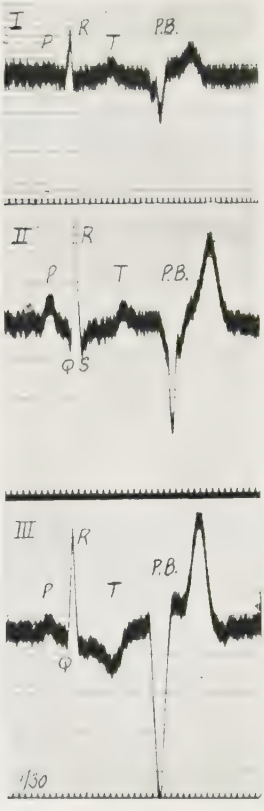


Fig. 173.

Fig. 172 and 173. Figures illustrating the two chief types of premature contractions of ventricular origin as they are portrayed in the separate leads in man. Fig. 172 shows a premature beat which arises in the right ventricle and Fig. 173 in the left ventricle. Figure 173 is atypical in that the deflections in lead *I* are usually reversed. Time in thirtieths of a second.

It is not difficult to choose from collections of curves, clinical and experimental examples showing close resemblances (Fig. 168 and 169 and Fig. 176 and 177). Nevertheless, anything more than approximate localisation of the spontaneous beats is impossible at the present time, for we possess experimental observations upon the dog only; and we know that the lie of the heart and the arrangement of the Purkinje strands in this animal differ materially from that in man.

The constant form of extrasystole in the electrocardiograms of a given clinical subject, from day to day, month to month, or even year to year, is very remarkable (*489*), and indicates the constancy of the focus of irritation.

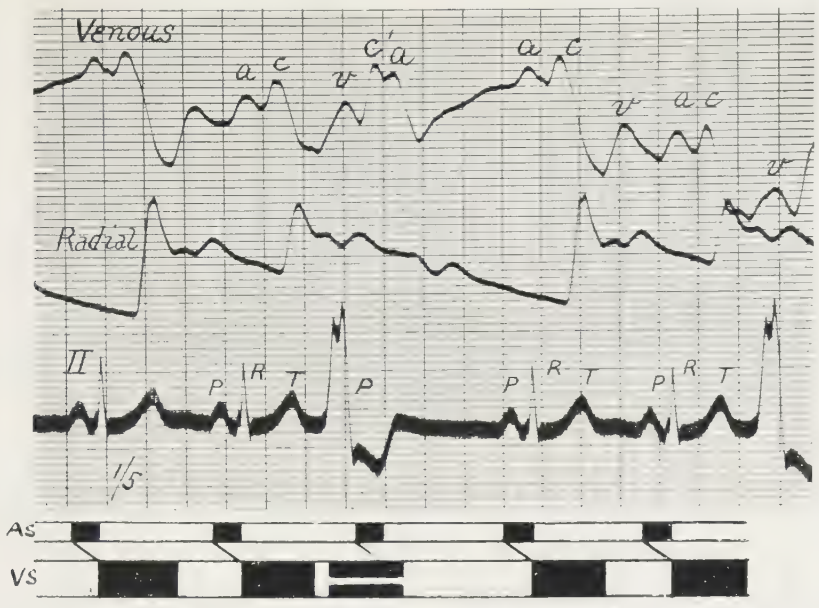


Fig. 174. Simultaneous venous, radial and electrocardiographic curves from a patient, showing an extrasystole arising in the right ventricle. A diagram placed below illustrates the mechanism of the heart over the period of the disturbance. Time in fifths of a second.

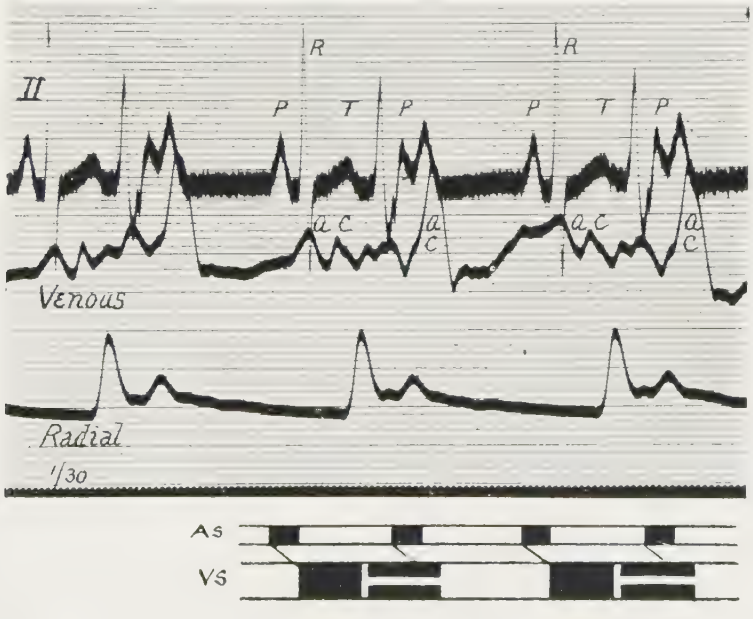


Fig. 175. Simultaneous electrocardiogram, venous and radial curve from a patient exhibiting a bigeminal action of the ventricle, resulting from ventricular extrasystoles. A diagram placed below illustrates the relation of chamber contractions. Time in thirtieths of a second.

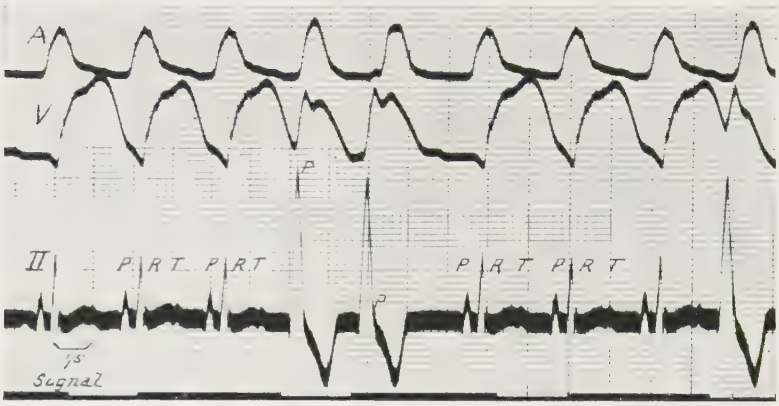


Fig. 176. ($\times \frac{4}{5}$.) Simultaneous curves from the auricular and ventricular muscle (*A* and *V*) and an electrocardiogram from a dog. The heart is responding from time to time to induction shocks thrown into the right ventricle. Time in fifths and twenty-fifths of a second.

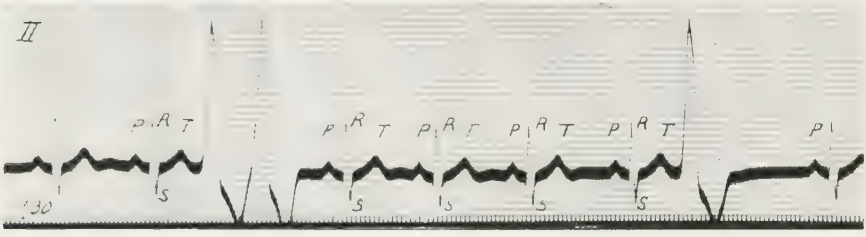


Fig. 177. ($\times \frac{4}{5}$.) A human curve for comparison with the Fig. 176. The disturbance of the heart's regular action is due to extrasystoles arising in the right ventricle. Time in thirtieths of a second.

It is relatively infrequent to find anomalous beats of more than one kind in a patient but, if found, both types are usually maintained. It has been suggested that these facts are to be explained by supposing the Purkinje system to be the chief source of such extrasystoles (*489*).

CHAPTER XVIII.

AURICULAR EXTRASYSTOLES.

THE premature contraction, when forced by stimulating the auricle, is followed by a similar premature contraction of the ventricle (Fig. 178). The disturbance affects both chambers, but the disorder is a little less in the ventricular than in the auricular movements. The conduction interval

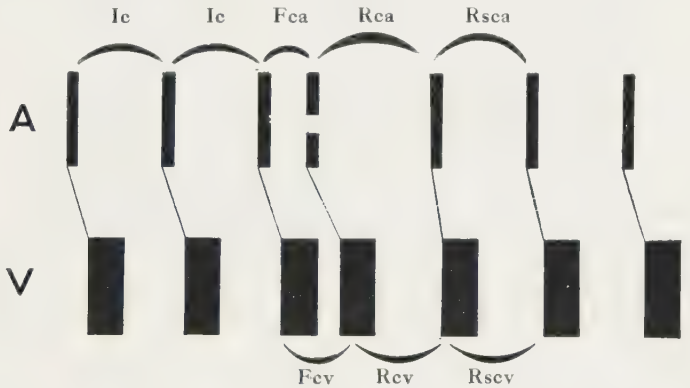


Fig. 178. Diagram of the events when the heart is disturbed by a premature auricular contraction. *Ic* = initial cycle; *Fca* = extrasystolic or forced auricular cycle; *Fcv* = extrasystolic or forced ventricular cycle; *Rca* and *Rcv* = returning auricular and returning ventricular cycles; *Rsca* and *Rscv* = restored auricular and restored ventricular cycles, respectively.

varies according to the degree of preceding rest, the *As-Vs* interval of the forced beat being prolonged and that of the returning beat shortened. These changes are illustrated in an exaggerated form in the diagram (Fig. 178); they may be conspicuous in clinical instances of premature contractions where there is primarily impaired conduction, but when the heart is normal they are inconspicuous and may require very precise measurement fully to display them (Fig. 181).

The returning cycle in the case of a premature auricular contraction is variable in length; it may be equal to an initial cycle, it may be much longer and compensatory, that is to say its duration taken with that of the forced cycle may be equal to two initial cycles or, finally and usually, its length may have an intermediate time value (*96*, *252*, *759*).

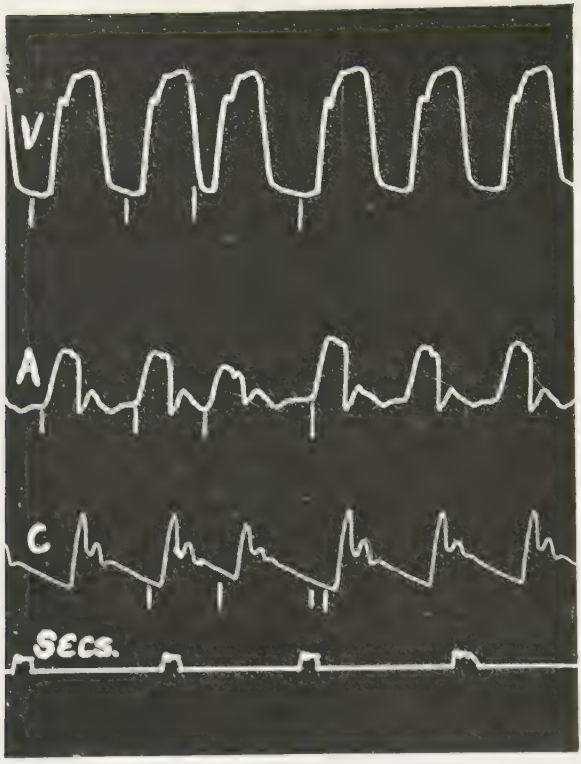


Fig. 179. Myocardiographic curves (A = auricle, V = ventricle) and Hürthle carotid pressure curve (C) from a dog. A single and spontaneous contraction of the auricle is shown; the ventricle follows suit and contracts early. The pause in the carotid curve is not fully compensatory. A curve taken from an experiment in which the right coronary artery was tied. [illegible] seconds.

Engelmann (*130*), in his researches upon the amphibian heart, found that when a premature beat is excited from the sinus, the returning cycle has exactly the same length as an initial cycle. It is supposed that the forced beat discharges the immature impulse and that this is built up again from the instant of its premature discharge and at the accustomed rate (Fig. 182). Stimulation of the mammalian pacemaker produces similar effects (Figs. 180 and 183) according to my own observations (*445*, *490*), and the recent experiences of Sansum (*687*). When this region is excited, the returning cycle does not exceed the initial cycle by greater time intervals than one or two hundredths of a second,* and often the two cycles may be of equal

* It is not easy to stimulate the actual pacemaker, for the node is a long structure and the rhythm springs from a point. Where there is this difference in the lengths of the cycles, it is probably due to inaccuracy in placing the stimulus. Hirschfelder and Exster (*316*), in the days before the pacemaker was located, were unable to find differences in the length of the returning cycle according to the point stimulated.

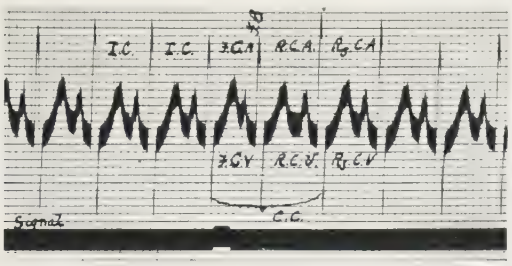


Fig. 180. (*Heart*, *1913-14*, *V*, *335*, *Fig. 1.*) Electrocardiogram from a dog showing a single premature contraction forced by stimulating the right auricle near the top of the sulcus. The lettering is the same as in Fig. 178. *F.B.* = forced beat. The initial cycle (*I.C.*) and returning cycle (*R.C.A.*) are almost equal in length. Time in twenty-fifths of a second.

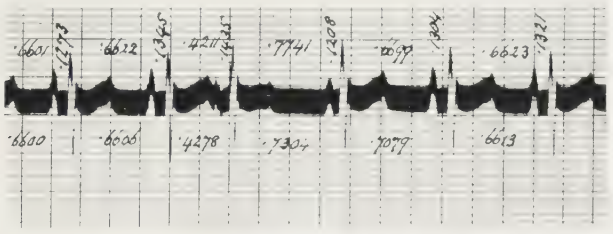


Fig. 181. (*Heart*, *1913-14*, *V*, *335*, *Fig. 2.*) Electrocardiogram from a dog. A single premature beat forced from the inferior caval region. The figures are time measurements, in decimals of a second, of the auricular (above) and ventricular cycles (below), and of the *P-R* intervals (up and down). Time in fifths of a second.

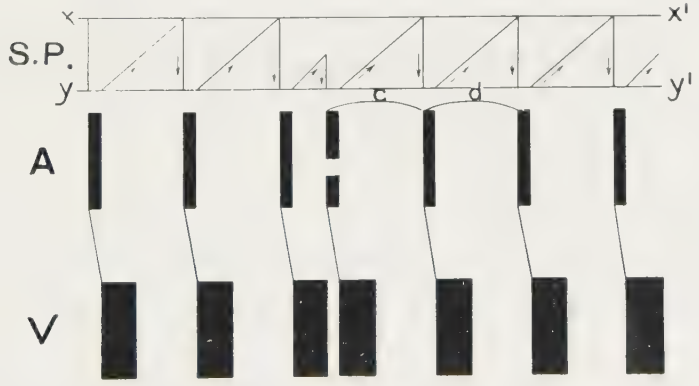


Fig. 182. A diagram illustrating the disturbance of the heart's mechanism when a premature contraction is excited in the neighbourhood of the pacemaker. Stimulus production in the tissue which originates the heart rhythm is indicated by the line *S.P.*; the impulse is supposed to explode when it reaches the line *x x'* and to fall at each contraction of the heart to the level *y y'*. *c* and *d* are equal in length.

length. It seems probable, therefore, that the time interval which elapses between the discharge of the pacemaker and the full growth of the next impulse is constant and independent of the manner in which the discharge is brought about; it seems to be the same if a stimulus arises in or is directly applied to the pacemaker, or if an excitation wave reaches the pacemaker from some other region of the auricle.

The length of the returning cycle exceeds the length of an initial cycle when the stimulus is applied to some outlying region of the auricle. It exceeds the initial cycle by the time taken for the excitation wave to travel to the pacemaker from the point of stimulation, as long since suggested (*96*, *759*), and recently demonstrated (*483*). The time interval between the forced beat and the returning beat—the returning cycle, as I term it—comprises the period during which, in the first place, the excitation wave is travelling from the point of stimulation to the pacemaker where it discharges the forming impulse, and during which, in the second place, the new impulse is being built.*

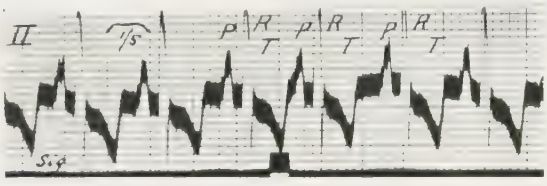


Fig. 183. Electrocardiogram from a dog showing a single premature contraction of the auricle, forced from the region of the pacemaker. The returning cycle in the auricle has the same length as the initial cycle and the complex *P* of the premature beat is normal in outline. Time in fifths and twenty-fifths of a second. The signal of stimulation is shown below.

When the returning cycle is compensatory, as happens rarely, then it is assumed that impulse formation in the *S-A* node has remained undisturbed, that is to say, the excitation wave of the premature beat has failed to reach the pacemaker. This may happen (*490*) if the forced beat comes relatively late in diastole, and the *S-A* node discharges its rhythmic impulse before the extraneous wave arrives; the two waves, natural and forced, then meet in the auricular walls† (Fig. 185 and 186).

* This method of forcing extrasystoles was at one time used in the attempt to isolate the mammalian pacemaker (*252*, *316*), but was unsuccessful, although the results which it yields do actually conform to the position of the pacemaker as it is now recognised.

† This explanation does not apply to all compensated disturbances of the human auricular rhythm; some are, I believe, accidental and attributable to temporary slowing of the heart.

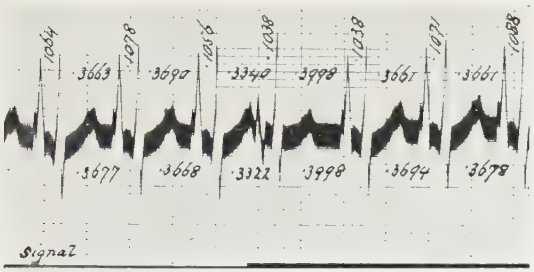


Fig. 184.

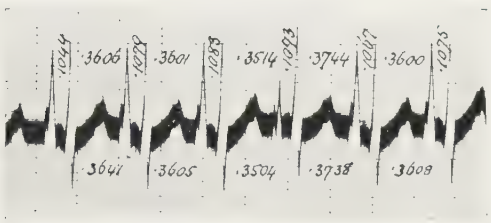


Fig. 185.

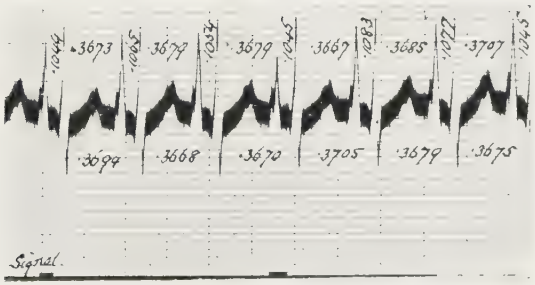


Fig. 186.

Fig. 184-6. (*Heart, 1913-14, V, 335, Fig. 5, 6, 7.*) From a dog. Three examples of a forced contraction disturbing *S-A* rhythm. The point of stimulation was in each instance the inferior cava. The shape of the pure inferior caval complex is shown in Fig. 184. In Fig. 185 and 186 there is almost exact compensation, and this has resulted because the forced contraction waves have failed to reach the pacemaker before its discharge. This fact is witnessed to by the transitional form of the electric complexes. In each case two excitation waves, one of *S-A* nodal and one of inferior caval origin have met in the walls of the auricle. Time in fifths and twenty-fifths of a second.

Records of auricular extrasystoles.

Premature auricular contractions, or auricular extrasystoles, were first believed to occur in the human subject by Wenckebach (*754*, *755*) and Cushny (*86*); these writers had arterial curves alone to guide them to their conclusion. Mackenzie's polygraphic records (*500*), and later electrocardiograms, have fully substantiated their view.

Arterial curves. — The arteriograms are similar in appearance to those disturbed by ventricular extrasystole, the premature beat is weak (Fig. 187) and may fail to appear in the curve. But measurement of the curves demonstrates a disturbance of the dominant rhythm of the heart (see page 132 and Fig. 187); the returning cycle is not compensatory except in rare instances.

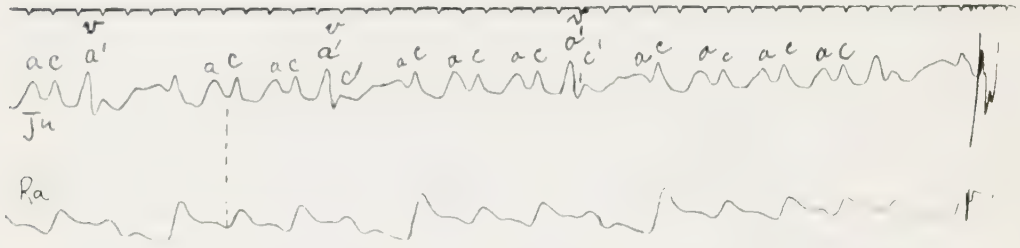


Fig. 187. A polygraphic curve from a patient exhibiting premature auricular contractions. The normal cycles are accompanied by *a*, *c* and *v* waves. The premature radial beats are represented in the venous curve by waves *c'*. Preceding the latter are prominent waves due to auricular systoles, $\frac{v}{a}$. The auricle contracts prematurely and during the last phase of the preceding ventricular systole.

Venous curves.—These show the auricular wave *a'* preceding premature ventricular waves *c'*, *v'* (Fig. 188). If the auricular contraction falls with the preceding ventricular beat, as frequently happens, then the *a'* wave is exaggerated (Fig. 187).

Electrocardiograms.—If the mammalian auricle is stimulated by means of single induction shocks and the responses of the heart are studied in electrocardiograms, it is found that the curves vary as each new region of the muscle is selected for stimulation. The ventricular phases are of constant form and usually of the same form as those accompanying the natural heart beat (page 60, Fig. 32), for the beats are all of supraventricular origin. It is the auricular complex which changes. When stimulation is near the pacemaker its outline is almost natural (Fig. 183). When stimulation is near the *A-V* node or inferior cava or the orifices of the pulmonary veins, the outline is often inverted or partially inverted (*445*).

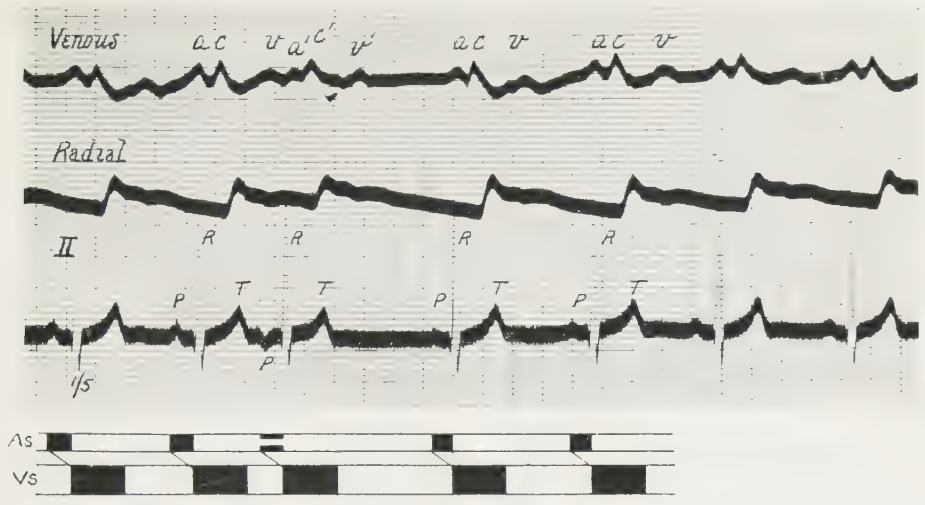


Fig. 188. ($\times \frac{4}{5}$.) Simultaneous venous, radial and electrocardiographic curves from a patient who presented auricular extrasystoles. A diagram representing the disturbed mechanism is placed below the photograph. Time in fifths of a second.

Auricular extrasystoles as they occur in man present the same features; the ventricular complex is usually of natural form, the auricular complex is variable in outline. Most commonly it is inverted in leads *II* and *III* (Fig. 188 to 190), clearly indicating the ectopic origin of the beat (*433*), and probably pointing to an origin from those portions of the auricle which are in the vicinity of the *A-V* groove. It may be upright, differing from the natural complex in minor detail; it may be isoelectric, and therefore invisible, (*451*) in some leads (Fig. 190, lead *I*). A beat arising in the auricle may be regarded as ectopic if its auricular complex departs from the natural complex in the same patient. It often happens, when an auricular extrasystole disturbs the heart's rhythm, that the anomalous auricular complex falls with the *T* of the preceding systole. In such cases the deflections are superimposed, *T* being double or notched (Fig. 189, compare T^1 and T^2 in the top curve), and the auricular systole is found by carefully comparing the outlines of the several *T* deflections of the curves.

The ventricular complexes associated with some premature auricular contractions are anomalous (*433*, *445*, *451*, *658*), and may be confused with the curves of ventricular extrasystoles. They should not be confused if they are preceded by clearly inscribed and premature auricular complexes, and the *P-R* interval is not shortened, for in these circumstances the auricle is indicated as the primary seat of irritation. The change in the form of the ventricular complex described is due, so it is maintained, to defects in conduction through some of the chief Purkinje strands (*445*), (*aberration*). The divergence of the ventricular complex from normality is most conspicuous when the extrasystole falls early in diastole; in some patients the degree

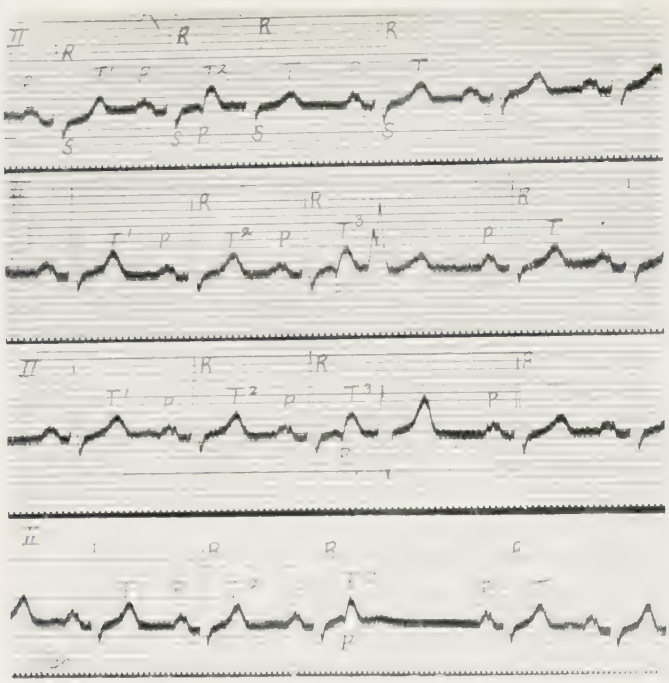


Fig. 189. Four curves from a single subject. Each shows a solitary premature auricular contraction. The premature auricular complex falls with the commencement of the preceding *T* and notches it. The corresponding ventricular complexes of the first three curves are of various forms; the central curves illustrate "aberration." In the last curve the premature auricular contraction is blocked. Time in thirtieths of a second.

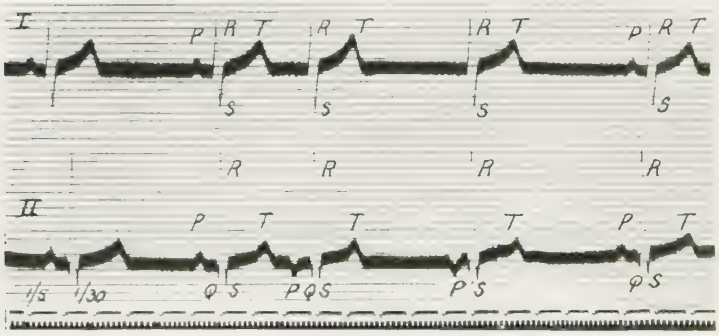


Fig. 190. Two curves (leads *I* and *III*) from the same patient and showing each an auricular extrasystole. In lead *I* the premature auricular complex is isolectric and therefore invisible. Time in fifths an thirtieths of a second

of aberration is closely connected with the degree of prematurity. It is notable that in these patients minor conduction defects in the main *A-V* bundle are the rule (see last curve of Fig. 189). In Fig. 189 two conspicuous forms of aberration, occurring in one and the same patient, are depicted. The last curve illustrates an auricular extrasystole which has failed to evoke a ventricular response, the blocked auricular extrasystole first described by Hewlett (*307, 433, 640, 655, 658*); the additional *P* deflection falls with T^3 and notches it and a long diastole follows. Aberration has been recorded in experiments as an accompaniment of forced auricular contractions; the form of the ventricular curve in these circumstances is independent of the site of auricular stimulation, but it is governed largely by the phase of diastole in which the stimulus falls; the distortion is greater if the stimulus falls in early diastole (*445*).*

Sinus extrasystoles.—As a rare phenomenon, extrasystoles occur in the human subject which are to be interpreted as arising in the *S-A* node; they have been termed *sinus extrasystoles* (*758*).

The returning cycle is no longer, it may be actually shorter, than the initial cycle (Fig. 191). In electrocardiograms the auricular summits are of constant outline, whether they belong to the rhythmic series or to the premature beats.

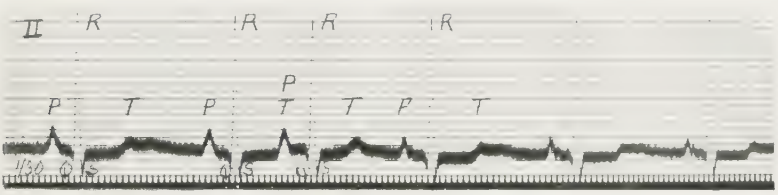


Fig. 191. Human curve. A single premature contraction, probably arising in the immediate neighbourhood of the sino-auricular node. The rhythmic and premature contractions of the auricle yield similar electric curves; the returning cycle is in this instance shorter than the initial cycle.

* Auricular extrasystoles, which yield ventricular complexes of aberrant types, may be forced experimentally by stimulating the auricle in an animal in which slight conduction defects have been induced already, for example in the cat by asphyxiation (unpublished observations).

Chapter XIX.

Premature beats arising in the junctional tissues; retrograde beats; premature beats disturbing new rhythms, etc.

Premature beats arising in the junctional tissues.

Examples of experimental extrasystoles (*269*) and of clinical extrasystoles (*303*, *512*), have been described* in which both the auricle and ventricle contract prematurely, and in which the contractions of auricle and ventricle begin almost simultaneously.

They are explained by supposing that the new impulse is formed in the junctional tissues, and that the excitation wave spreads at one and the same time to auricle and ventricle (Fig. 192). Contraction of the auricle in response to one impulse and of the ventricle in response to a distinct impulse cannot be allowed, for in patients who present these singular beats, the same events are exactly repeated.

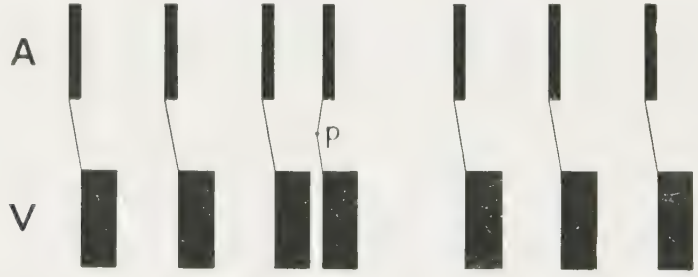


Fig. 192. A diagram illustrating the mechanism of the heart when the sequence is disturbed by a premature beat arising in the junctional tissues.

A clear example of this disturbance of the heart's action is to be found in the accompanying figure, which illustrates the features of these beats in simultaneous records (Fig. 193). In the arterial curve of this example, the disturbance resembles the premature ventricular contraction in every respect, for the returning cycle is compensatory ; in other examples the cycle

* The first clinical examples are those published as retrograde contractions by Pan (*585*), and by Volhard (*741*).

is less than compensatory in its length. The venous curve shows a tall wave $\frac{a}{c}$, resulting from contraction of the auricle while the tricuspid valve was closed; a prominent wave of similar origin may be found as an accompaniment of ventricular extrasystoles, but the junctional extrasystole is distinguished by the position of the composite wave; it falls before the time when the rhythmic auricular wave is expected. In the case of the ventricular extrasystole, the exaggerated *a* wave belongs to the rhythmic series; its coincidence with ventricular systole is in this circumstance accidental; in the case of the junctional extrasystole the auricular rhythm is disturbed and coincidence is forced. In the electrocardiogram a premature ventricular complex is seen and this is of sufficiently normal outline to show that the ventricle is stimulated from a supraventricular focus; the auricular complex is buried and hidden in the ventricular complex and in this instance is not clearly seen; the position of the auricular systole may be judged by comparing the venous and electrocardiographic curves.

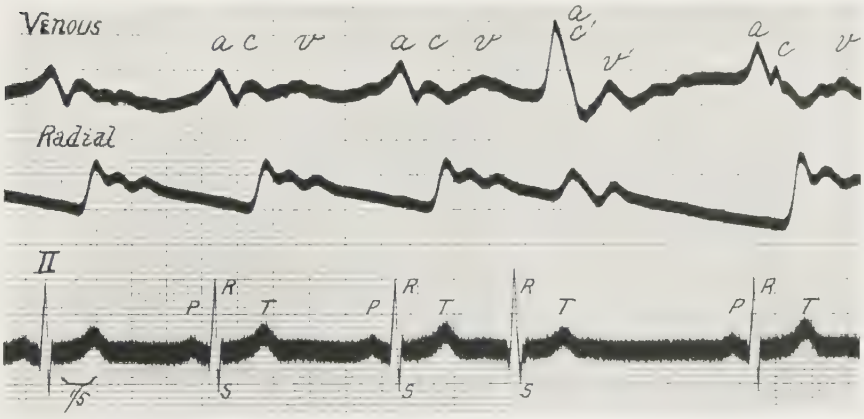


Fig. 193. ($\times \frac{9}{10}$.) Venous, radial and electrocardiographic curves from a patient who exhibited extrasystoles arising in the region of the *A-V* node. Time in fifths and twenty-fifths of a second.

In other instances of premature beats, whose origin is ascribed to the junctional tissues, the auricle contracts before the ventricle; the *P-R* interval, however, is slightly or conspicuously shortened; in these curves a greater or lesser part of the auricular complex is visible and the origin of the beat from the junctional tissues is confirmed by the inversion of *P*. In yet other instances, there is a distinct *Vs-As* interval. An example of this class is shown in Fig. 194. Somewhat less than one-fifth second after the beginning of the first premature ventricular systole of this figure, the auricle also contracts, and the inverted auricular complex, (*P*) marked below the curve, notches the beginning of *T*. The origin of this extrasystole is clear. The

chambers have contracted in response to a common impulse, for in both chambers contraction is premature. The contraction of the ventricle precedes that of the auricle by an interval which is rather less than the natural *P-R* interval in the same subject; the impulse has arisen at such a point that the spread to the ventricle takes less time than the spread to the auricle. The impulse has not arisen in the ventricle itself, for the shape of the ventricular complex indicates the supraventricular origin of this beat. The inversion of *P* corresponds to the abnormal course which the excitation wave takes in the auricle. The shape of the auricular complex is consistent with the spread through the auricle from the *A-V* node; and the length of the *Vs-As* interval indicates the origin of the impulse from the lower reaches of the *A-V* node or the *A-V* bundle (*477*).

When the extrasystole is less premature, it may happen, as in the second premature beat of the same figure, that, while the impulse is passing towards the auricle, the auricle responds to the rhythmic impulse of the *S-A* node (*448*). The nodal impulse finds the auricle in the refractory state and the natural outline of the auricular contraction (the seventh *P* of the curve) is consequently maintained.

Retrograde beats.

In some rare instances where auricle and ventricle both contract prematurely and where a *Vs-As* interval is found, it is probable that the extrasystole has arisen in the ventricle and has spread in retrograde fashion to the auricle.

That the heart's mechanism may be reversed, *i.e.*, that the auricle may beat in response to the ventricle, is readily shown by stimulating the ventricle with successive induction shocks sent in at a rate exceeding the previous heart rate. The ventricle responds to each stimulus and, after a variable number of cycles, each ventricular response is followed by an auricular contraction. The impulses are conveyed from the beating ventricle to the auricle through the *A-V* bundle. But it is generally acknowledged (*26*, *523*), that in most animals conduction from ventricle to auricle takes place less readily than from auricle to ventricle; and it is usually considered that the interval *Vs-As* of retrograde beats is longer than is the *As-Vs* interval of the normal heart beats in the same animal.*

* This is, I think, open to question; the difference is in any case inconsiderable and the measurements of the intervals are beset with sources of small error, whether they are calculated in myocardiograms or electrocardiograms. The difficulty of exact calculation comes in estimating the beginning of contraction in a heart chamber; the myocardiogram records contraction of the particular muscle area to which it is attached; the electrocardiogram employed at ordinary sensitivities and taken from a limb lead does not signal the earliest involvement of the muscle. Although this doubt as to the actual intervals exists, yet there is little doubt that retrograde conduction is more easily hindered or disturbed than is forward conduction. In the presence of a slight defect of conduction in the *A-V* tissues, a retrograde action cannot be induced by rhythmic stimulation of the ventricle (*484*).

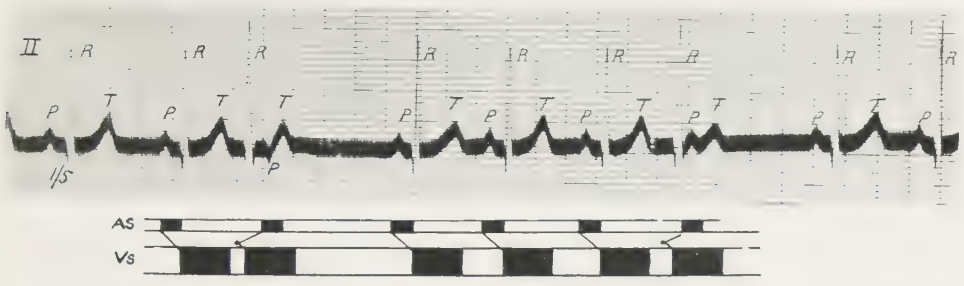


Fig. 194. ($\times \frac{4}{5}$.) An electrocardiogram from a patient who exhibited extrasystoles arising in the junctional tissue. The first extrasystole involves both ventricle and auricle; the ventricular complex is of the supra-ventricular type; the auricular complex is inverted and notches T. The second extrasystole involves the ventricle only; it falls later in diastole and the A-V nodal impulse finds the auricle refractory, as the latter is already contracting in response to a S-A nodal impulse. Time in fifths of a second.

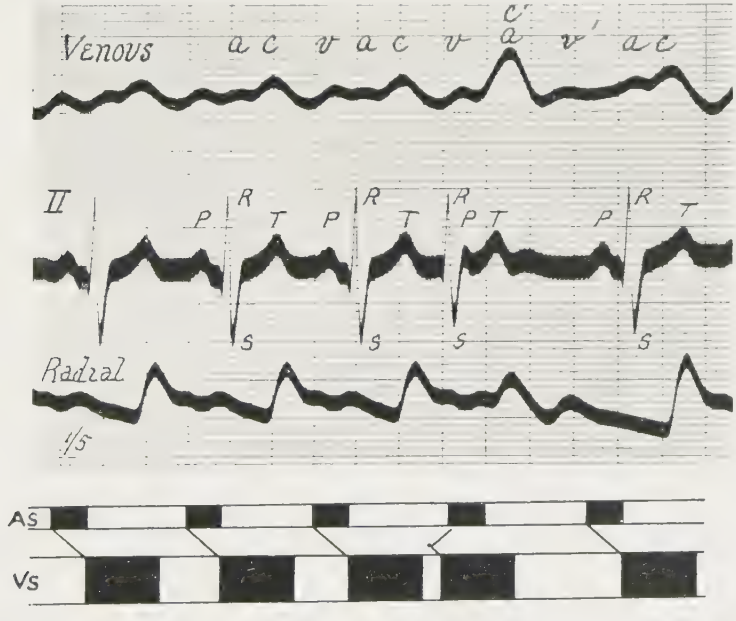


Fig. 195. Simultaneous venous, electrocardiographic and radial curves, showing an extrasystole arising in the junctional tissues. The ventricle responds to the new impulse, the auricle does not, for it is already contracting in response to the S-A nodal impulse. Time in fifths of a second.

But the relation of these two intervals and the factors governing reversed conduction are still by no means clear. That an impulse from a premature ventricular contraction is not conveyed to the auricle as a rule is easily understood, for at the time when the reversed impulse should reach the auricle that chamber is in contraction in response to the *S-A* node and is therefore refractory to further stimulation. But a similar explanation does not hold good for all premature contractions arising in the ventricle; it is not always clear for example why some ventricular extrasystoles are interpolated and yet fail to reach the auricle (see footnote page 207).

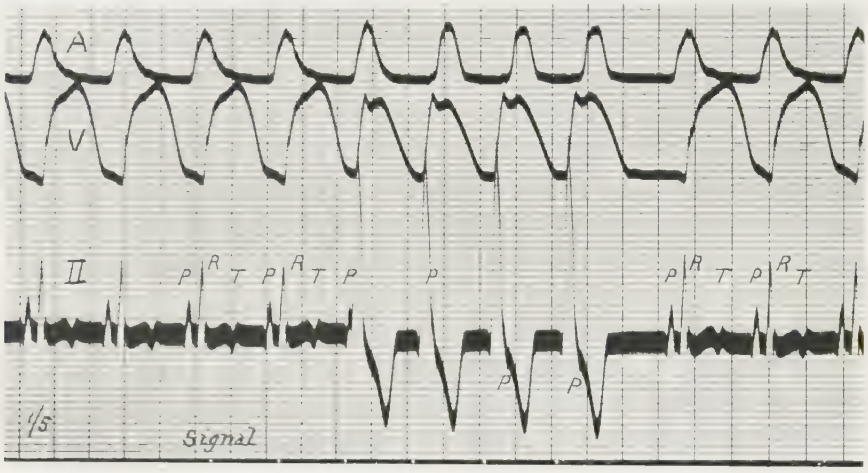


Fig. 196. ($\frac{9}{10}$.) Myocardiograms from auricle (*A*) and ventricle (*V*) and electrocardiogram from a dog. The normal rhythm is disturbed by four rhythmic beats forced from the conus region of the right ventricle by rhythmic stimulation (the signal marks of stimulation are white dashes at the bottom of the photograph). For two cycles the auricle fails to respond to the forced ventricular beats, the impulses conveyed from the latter falling during the refractory period of the auricle. The last two cycles show response of the auricle (see myocardiogram), and the corresponding auricular complexes are minute notches (*P*) on the ventricular complexes; these little notches are more distinct in Fig. 171 (page 216), which is from the same animal. Time in fifths of a second.

Pan (*585*) and Volhard (*741*) explained certain clinical curves on the assumption that the extrasystoles arose in the ventricle and were retrograde to the auricle, but it seems more probable that the examples with which they dealt were junctional extrasystoles. I have seen but a few examples of what may be regarded as isolated retrograde extrasystoles in man.*

* The reversed rhythm described by Williams (*780*) is an undoubted example of a rhythm arising in the junctional tissues, for the ventricular complexes are of supraventricular type. A similar explanation might be applied to the case recorded by Norrie and Bastedo (*577*), though in this instance the mechanism is uncertain; their published curves might be interpreted in either way or might be attributed to an unusually prolonged *a-c* interval.

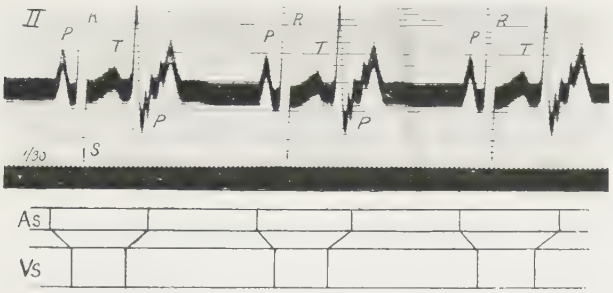


Fig. 197. ($\times \frac{11}{15}$.) Electrocardiogram from a subject who presented ventricular extrasystoles. From time to time these extrasystoles were retrograde to the auricle as in the present figure. The opening of each abnormal ventricular complex is followed by an abnormal *P* (consisting of two notches). Compare Fig. 175, page 219, which, though from the same case, shows no retrogression. The present figure has been somewhat reduced from its original size. Time in fifths of a second.

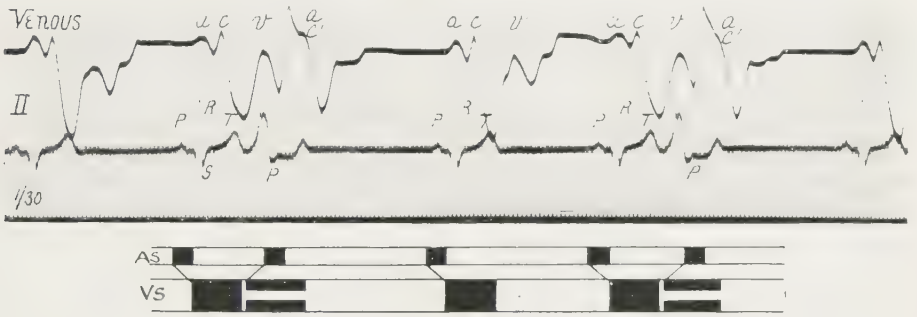


Fig. 198. ($\times \frac{9}{10}$.) Simultaneous venous curve and electrocardiogram from a patient who exhibited what were in all probability retrograde extrasystoles. Each normal cycle of the heart is followed by a premature beat of the ventricle. This complex is abnormal. An auricular systole, also premature, follows a little later; it is clearly exhibited in the jugular curve, and appears as a small dip in the electrocardiogram. Time in thirtieths of a second.

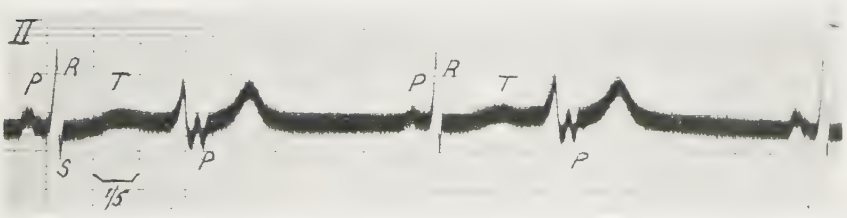


Fig. 199. A bigeminal action of the heart probably due to retrograde beats arising prematurely in the ventricle. The premature ventricular complex is anomalous and is notched by an inverted *P*. Time in fifths and twenty-fifths of a second.

An example is shown in Fig. 197. Each normal cycle is followed by an abnormal ventricular complex, buried in which is an anomalous auricular complex *P*. The excitation wave has taken an abnormal course through *ventricle* and auricle, the former chamber leading. The curve is from the same patient as that of Fig. 175, page 219, with which it should be compared. A second possible example of retrograde beats is shown in Fig. 198. Here the premature *P* is not very clearly defined in the electrocardiogram, but the large premature wave $\frac{a}{v}$ is distinct in the jugular tracing. A third possible example is seen in Fig. 199.

But while these examples show the expected features of retrograde beats, it must be stated that another explanation may be applied to them. The premature beats may be of *A-V* nodal origin ; in that case the abnormal ventricular complex would be supraventricular, but aberrant. In the case of the example shown in Fig. 197 this last explanation is most unlikely, for in this patient the type of ventricular complex was constant from week to week, and constancy is not a feature of (extrasystolic) aberrant contractions.

The form of the auricular complex corresponding to retrograde beats is discussed in Chapter XXI.

Ventricular extrasystoles and complete heart-block.

If auriculo-ventricular dissociation is brought about in the heart of an animal by destroying the *A-V* bundle,* and the slow ventricular rhythm is subsequently disturbed by a premature beat forced by stimulating the ventricle, then the returning cycle is of approximately the same length as the cycles of the idio-ventricular rhythm (*265*). This relation of the cycles in the ventricle is similar to that observed in the auricle when an extra beat is forced from the *S-A* node, and a similar explanation is customary. It is supposed that the forced beat destroys the immature impulse which is forming in the ventricle and that a new impulse is built up at the same point and at the usual rate. This hypothesis is expressed diagrammatically in Fig. 200. As it stands at the present time the hypothesis is not entirely satisfactory. The idio-ventricular rhythm has its origin in the *A-V* bundle below the lesion ; the ventricle is stimulated at its surface ; it might be anticipated that the duration of the returning cycle would be that of an idio-ventricular cycle, *plus* the interval taken for the excitation wave to travel from the point of stimulation to the centre of impulse formation, and the last interval is appreciable.†

* The results are not the same when *A-V* block is produced by poisoning the heart (*669*). The returning cycle may then be compensatory ; presumably because the impulse centre is in the *A-V* node and not in the bundle.

† The observation should be repeated, the electrocardiograph being employed as recorder. The intervals as measured in mechanical curves are open to too considerable an error.

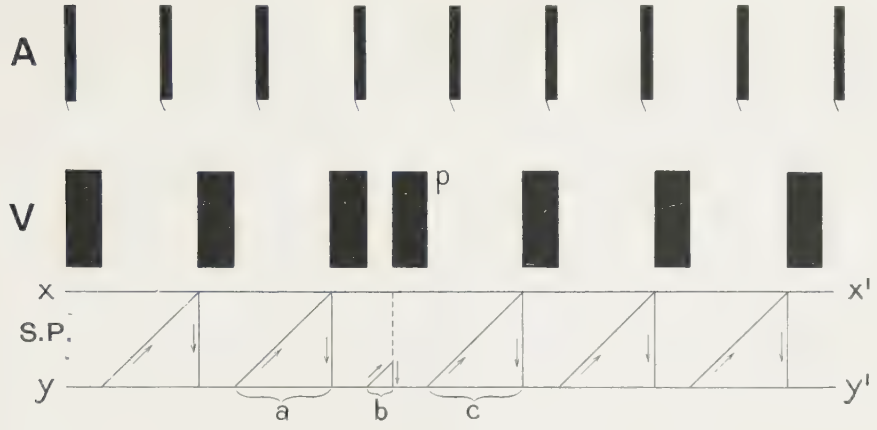


Fig. 200. A diagram illustrating the events when an extrasystole arises in a ventricle which is dissociated from the auricle by section of the *A-V* bundle. The vertical rectangles *A* and *V* represent contractions of auricle and ventricle respectively. The oblique lines placed below (*S.P.*) represent stimulus production in the ventricle: the impulses are represented as built up at a uniform rate, and as discharged with each ventricular systole to which they give rise. The point of discharge lies at the line *x*, *x'*. The premature beat *p* destroys the stimulus material which has accumulated over the short interval *b*. The next systole follows a diastole (*c*) which is equal to the diastole (*a*) of the regular spontaneous rhythm.

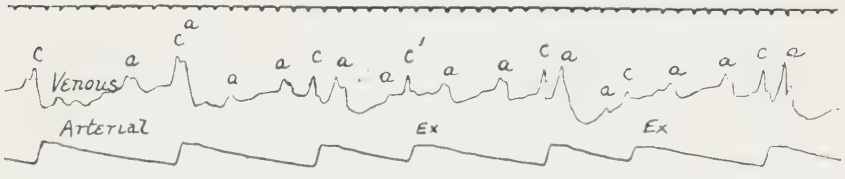


Fig. 201*a*. Venous and radial curves from a patient who exhibited permanent and complete *A-V* heart-block. Two extrasystoles of ventricular origin are also seen. The returning cycles are equal in length to the initial cycles.

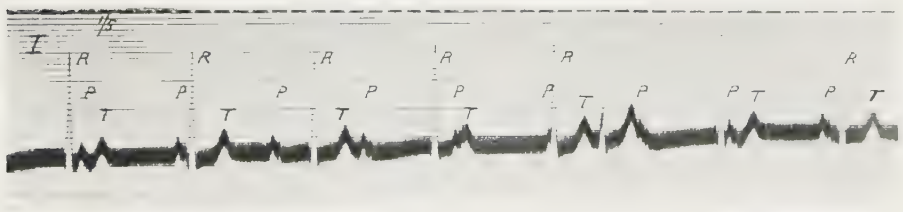


Fig. 201*b*. ($\times \frac{11}{15}$.) An electrocardiogram from a patient. Complete *A-V* heart-block is present and the ventricular rhythm is disturbed by a ventricular extrasystole. The length of the returning cycle is precisely that of the initial cycles. Time in fifths of a second.

Clinical examples of idio-ventricular rhythm disturbed by spontaneous ventricular extrasystole are seen in Fig. 201*a* and *b*. In each of these figures the returning cycles and the initial cycles have precisely the same duration.*

When auricular extrasystoles occur as a complication of complete heart-block, the regular rhythm of the ventricle is undisturbed, while the events in the auricle are similar to those seen when the same form of extrasystole interrupts a normal *S-A* rhythm.

Atrio-ventricular rhythm and extrasystoles.

If the heart is beating in response to a rhythm arising in the *A-V* node, and solitary contractions are forced in the auricle (*302*, *490*, *669*), the events are as they are seen in Fig. 202 and 203. When as in Fig. 202 the forced auricular beat occurs early in diastole, it is responded to by the ventricle.

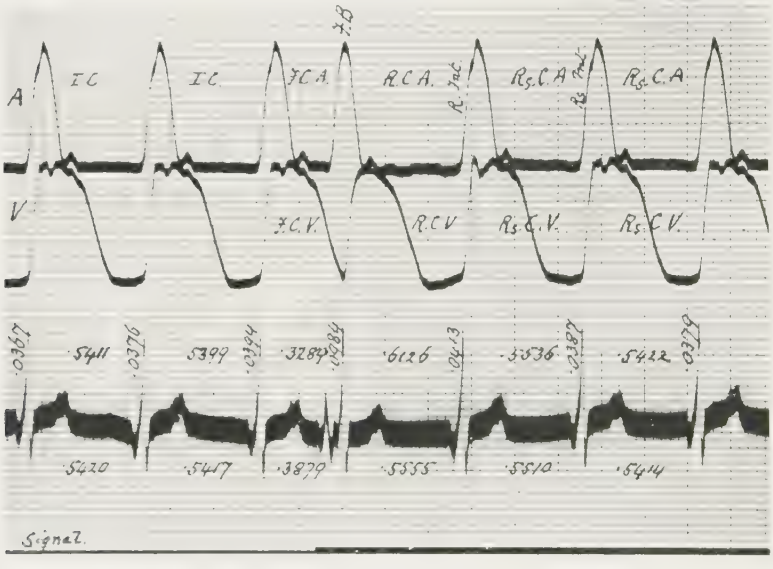


Fig. 202. (*Heart, 1913-14, V, 335, Fig. 3.*) Simultaneous myocardiograms and electrocardiograms from a dog, showing a forced beat of inferior caval origin disturbing *A-V* rhythm. In the electrocardiogram the beginning of the inverted *P* is seen immediately before the corresponding ventricular complex. *I.C.* = initial cycles. *F.C.A.* and *F.C.V.* = forced cycle (auricular and ventricular). *F.B.* = forced beat. *R.C.A.* and *R.C.V.* = returning cycles. *Rs. C.A.* and *Rs. C.V.* = restored cycles. *R. Int.* = returning *As-Vs* interval. *Rs. Int.* restored *As-Vs* interval. The lengths of the auricular and ventricular cycles and of the *As-Vs* intervals, are given in decimals of a second. The auricle responds to the make shock of stimulation. Time in fifths and twenty-fifths of a second.

* This is not always the case in clinical curves ; sometimes the returning cycle is much shorter than the initial cycle (*463* (*Fig. 33*) *and 570*). The explanation of these exceptional curves is lacking.

The impulse travels through the *A-V* node, where the rhythm is consequently disturbed. The returning ventricular cycle is not compensatory, but has the length of an initial cycle or is a little longer or a little shorter than the initial cycle. The *A-V* rhythm is resumed after the disturbance, but for a

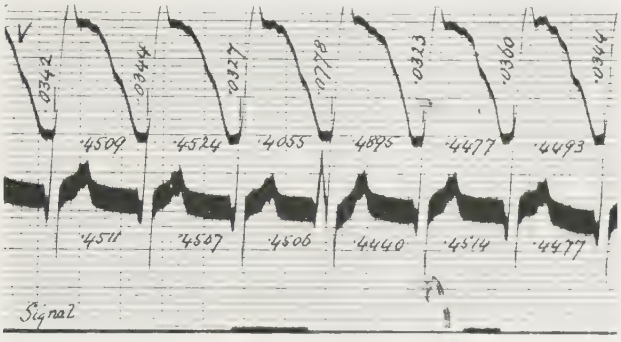


Fig. 203. (*Heart, 1913-14, V, 335, Fig. 8.*) *A-V* rhythm in a dog, disturbed by an extrasystole forced by a break shock from the right auricular appendix. From the same animal as the last. The forced beat comes late in diastole; it comes too late to disturb the rhythmic nodal impulse, to which the ventricle consequently responds. The returning ventricular cycle is therefore practically compensatory. The second break shock falls at a time when the auricle is refractory. Time in fifths and twenty-fifths of a second.

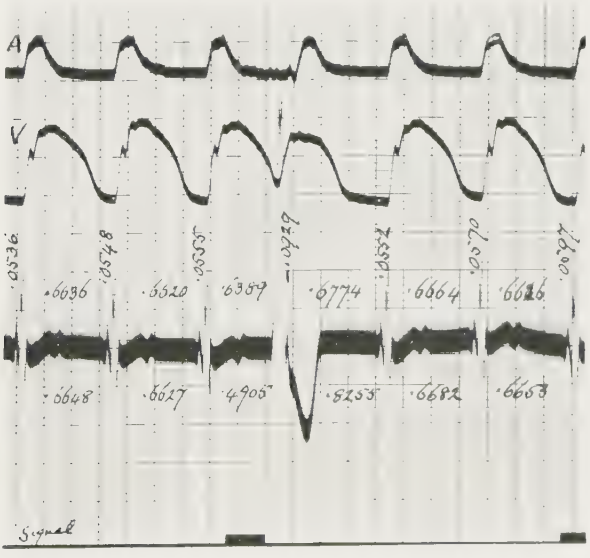


Fig. 204. (*Heart, 1913-14, V, 335, Fig. 12.*) *A-V* rhythm in a dog, disturbed by an extrasystole forced from the ventricle. The rhythm of the node is disturbed, as is shown by fine measurement; the auricle responds to the forced ventricular contraction. Time in fifths of a second.

few cycles the *As-Vs* intervals alter. When these intervals are short in the initial cycles, the returning interval (*R. Int.*) and first restored interval (*Rs. Int.*) is lengthened (Fig. 202).

If the forced auricular contraction falls late in diastole (Fig. 203) there is no ventricular response to it; the ventricle responds to the rhythmic nodal impulses and the disturbance is compensated.

When the premature beat is forced from the ventricle, similar events are observed, though the order of chamber contraction is reversed. Thus, if the forced ventricular beat comes early in diastole, the auricle responds and the returning auricular cycle has almost the same duration as an initial cycle (Fig. 204); while, if the forced beat is less premature, the rhythm of the node is undisturbed and the auricle responds to it regularly throughout (Fig. 205).

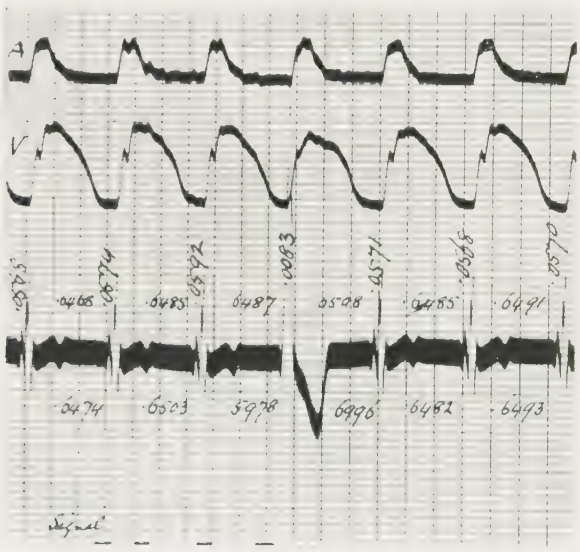


Fig. 205. (*Heart, 1913-14, V, 353, Fig. 11.*) *A-V* rhythm in a dog. A single beat has been forced from the ventricle late in diastole. The auricle does not respond to it, but to the rhythmic nodal impulse, and the disturbance is therefore compensated. The first three stimuli, as shown by the signal, were without effect. Time in fifths of a second.

Clinical examples of extrasystoles disturbing *A-V* rhythm have been recorded on rare occasions (*337, 434, 766*).

Chapter XX.

Paroxysmal Tachycardia of Supraventricular Origin.

Periodic acceleration of the heart's rate, a frequent clinical phenomenon, has its origin in many different causes. Sudden accessions of the rate at which the pace-maker elaborates impulses occur under purely physiological conditions, for example under the influence of emotion or during exercise. And in particular types of patients, more especially those who exhibit instability of the nervous system, a slight excitant may lead to tachycardia accompanied by sensations of distress; a conspicuous and unexpected response of the heart, in the form of increased rate, to relatively slight disturbance is also found where the body is invaded by organisms locally or generally, in exophthalmic goitre and many other conditions.

But apart from accelerations of these and similar natures, a specific type of tachycardia exists in the human subject, which demands separate and careful consideration. Often a grave malady, irremediable and occasionally directly fatal, it should be strictly isolated when its pathology is studied. It is usually characterised by the severity of its symptomatology, the abrupt onset and cessation of the attacks, and by the seemingly haphazard manner in which the crises are provoked or are assuaged. Treated from the pathological standpoint and as a tachycardia, it stands as a definite entity.

The general features of paroxysms of tachycardia are well illustrated by those paroxysms which arise in the auricular portions of the heart. Paroxysmal tachycardia is an affection in which from time to time and for variable periods the natural heart rhythm (*S-A* nodal rhythm) becomes submerged, and the heart responds to impulses formed at a more rapid rate in some other portion of its walls. If the exposed auricle is submitted in experiment to successive electric shocks and these shocks follow each other at a rate which exceeds that of the normal rhythm, the auricle beats in response to these shocks and, the ventricle following, the rhythm of the whole heart is controlled by the stimuli. The first response of the auricle is premature, and the new rhythm is maintained so long as the shocks continue. When these cease, a pause supervenes (*431*), the length of the last cycle bearing similar relations to that of a natural beat, as does the length of the returning cycle to that of an initial cycle in the case of an auricular

Fig. 206. ($\times \frac{2}{3}$.) Myocardiographic (A = auricular, and V = ventricular contraction) and electrocardiographic curves from a dog. The first four beats of the figure are natural systoles; following upon them are five responses of the heart to rhythmic electrical stimulation of the right auricular appendix (see signal); the last three cycles belong to the natural rhythm restored after a pause at the end of stimulation. The stimuli are signalled by the small white gaps in the black band at the bottom. Time in fifths of a second.

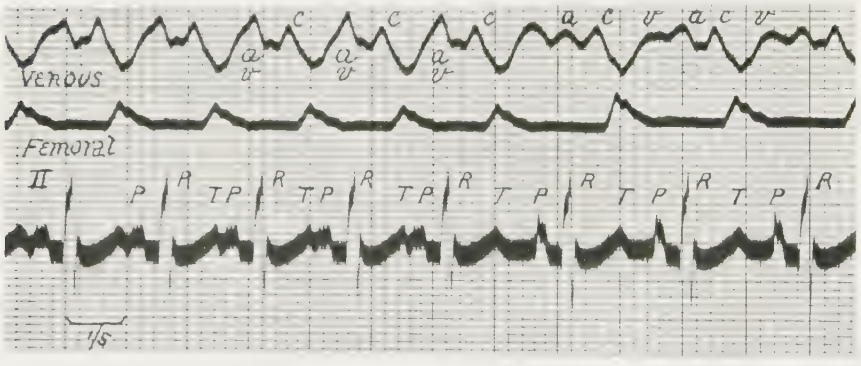


Fig. 207. Venous, carotid and electrocardiographic curves from a dog. To the left the beats are responses to stimulation of the right auricular appendix; to the right the beats are natural. Time in fifths and twenty-fifths of a second.

extrasystole (*431*, *433*). After the pause the natural rhythm is resumed and, if the heart is in good condition, it is resumed at precisely its original rate.

Paroxysms of tachycardia arising in the auricle in clinical subjects show similar features. They start abruptly, the first beat being premature (Fig. 208). The accelerated heart rate is regular and ends abruptly in a post-paroxysmal pause, following upon which the natural rhythm recommences.

Fig. 208. (*Agassiz. Heart, Vol. III, page 195, Fig. 1.*) Venous and radial curves showing the end of a paroxysm of tachycardia arising in the auricle, the resumption of the normal rhythm and the further onset of a new paroxysm.

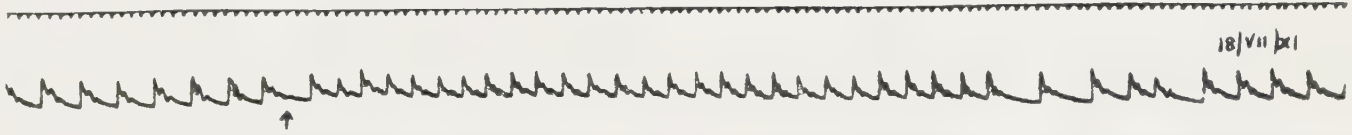


Fig. 209. (*Heart, Vol. III, page 180, Fig. 5.*) An arterial curve showing the onset and cessation of a short paroxysm of tachycardia of auricular origin. The first beat of the paroxysm was too weak to affect the pulse. Its position is marked by an arrow.

In clinical examples, the rate of the natural rhythm, before and after the paroxysm is the same in most instances. In exceptional cases the full rate of the natural rhythm is developed slowly at its resumption; in these the post-paroxysmal pause (or returning cycle) is of unusual length and the heart rate accelerates for six or more beats until the original rate is restored. The slow rate of the natural rhythm when this is resumed is comparable to the slow rate of the idio-ventricular rhythm when the latter has been interrupted by a forced tachycardia; but in the case of an *S-A* rhythm the retardation appears to be exhibited by pathological hearts only.

Fig. 210. (· ⅓) An arterial curve showing the termination of a paroxysm of tachycardia. The post-paroxysmal pause is unusually long and the natural rhythm at its resumption is slow but accelerates as it proceeds.

The action of the heart during the paroxysm is remarkable for its regularity, the beats follow each other at equal intervals; the regular sequence of the beats is conditioned by their origin from a single focus (as ascertained electrocardiographically). The rapid rhythm is also remarkable for the relative constancy of its rate for long periods and under various conditions; thus the rate is uninfluenced by posture and by exercise; it cannot be *retarded* by pressure on the vagi, though the new rhythm is sometimes abolished by this procedure (*64*). In short, these paroxysmal rhythms are not under the adjusting control of the central nervous system, as is the normal rhythm (*454*).

Reaction of rapidly beating hearts to postural changes of the body.

Simple Tachycardia.

	Standing.	Lying.		Standing.	Lying.
1. Pulmonary tuberculosis	142	133	2. Exophthalmic goitre	180	145
	144	130		176	147
	143	129			

Paroxysmal Tachycardia.

	Standing.	Lying.		Sitting.	Lying.
1. Cardiac case	205	213	2. Cardiac case	195	190
	209	204		200	199
	204	210		197	203
	212	212		199	187

The duration of paroxysms in a given patient is fairly constant; from subject to subject the duration is very variable. A single paroxysm may consist of six or more beats; it may continue for an hour, a day, a week or more, without interruption. Exceptionally in one and the same patient short and long paroxysms may be witnessed.

That which constitutes a paroxysm of tachycardia is a matter of terminology. The solitary extrasystole has been studied in a previous chapter, but extrasystoles may be grouped (*585*), the groups comprising two (Fig. 211), three or more beats (Fig. 214), and no dividing line can be drawn between these disturbances and such short paroxysms as are shown in Fig. 212.

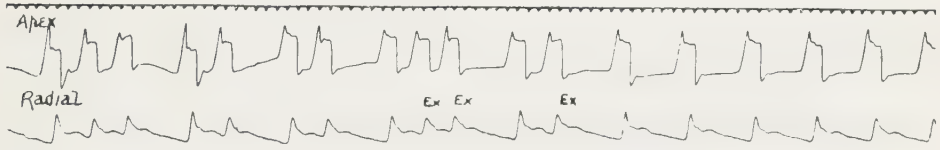


Fig. 210. ($\times \frac{1}{2}$.) An irregularity of the heart, resulting from single extrasystoles of auricular origin, and pairs of extrasystoles. Apical and radial curves are shown.

It is indeed notable that short paroxysms often immediately precede the onset, or immediately succeed the ending (Fig. 214) of a long continued paroxysm, and that in most patients who exhibit long or short paroxysms isolated premature contractions interrupt the slow periods of normal heart action. The isolated extrasystole and the shorter preliminary or terminal paroxysms have for the most part precisely the same origin as the long continued paroxysm in the same case (*433*); this fact has been determined repeatedly both by polygraphic and electrocardiographic analysis.

The point of origin.—In localising the seat of disturbance in patients who suffer from paroxysms of tachycardia, the same methods are adopted as for the extrasystole. The paroxysm may arise in auricle, *A-V* node or ventricle. The origin in some patients is readily determined by means of venous curves; it may be localised more exactly in most cases by the employment of the electrocardiograph.

Auricular origin.—A number of paroxysms are frankly auricular in origin (*55*, *80*, *171*, *243*, *431*, *433*, *655*), each paroxysmal beat being represented in the venous curves by *a* and *c* waves, though the former often falls with the preceding *v* wave and is of increased amplitude (Fig. 213). The same origin may be ascertained electrocardiographically, for the beats of such paroxysms present ventricular complexes of supraventricular type (*433*), and these are preceded by auricular complexes of anomalous outline, indicating the ectopic origin of the corresponding auricular impulses (*433*).

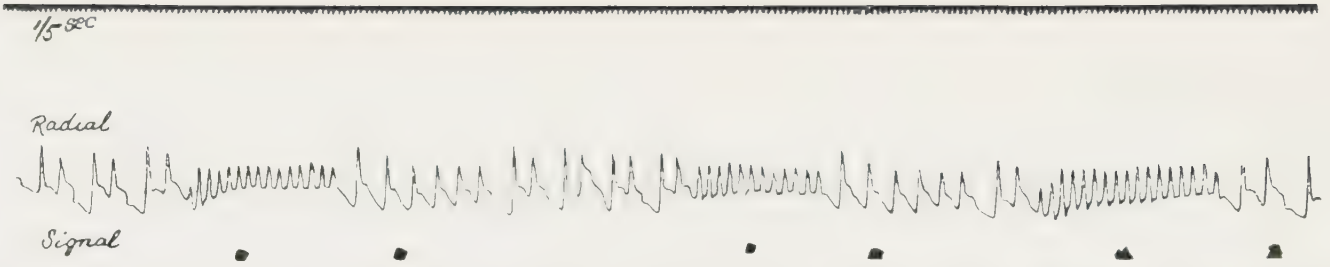


Fig. 212. (*Heart, 1909-10, I, 265.*) Radial curve from a case of paroxysmal tachycardia. Showing three short paroxysms, the onsets and endings of which were signalled by the patient. The slow periods are irregular, being interrupted by extrasystoles. These extrasystoles and the individual beats of the paroxysms were of a similar nature. They all arose at a single point in the auricular substance, removed from the pacemaker, as was ascertained electrocardiographically.

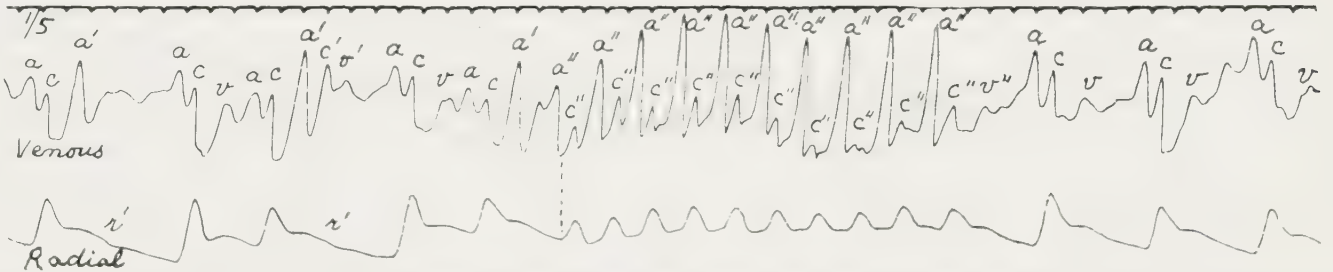


Fig. 213. (*Heart, 1909-10, I, 265.*) Polygraphic curve taken at a faster rate from the same patient. Showing that the interruptions of the slow periods are premature auricular contractions, and that the paroxysms are of similar nature.

In this curve and in the former the paroxysms end in the characteristic post-paroxysmal pauses. Where the onset is shown, it is abrupt; there is neither a gradual transition from fast to slow nor slow to fast rate, neither is there a simple mathematical ratio between fast and slow rates.

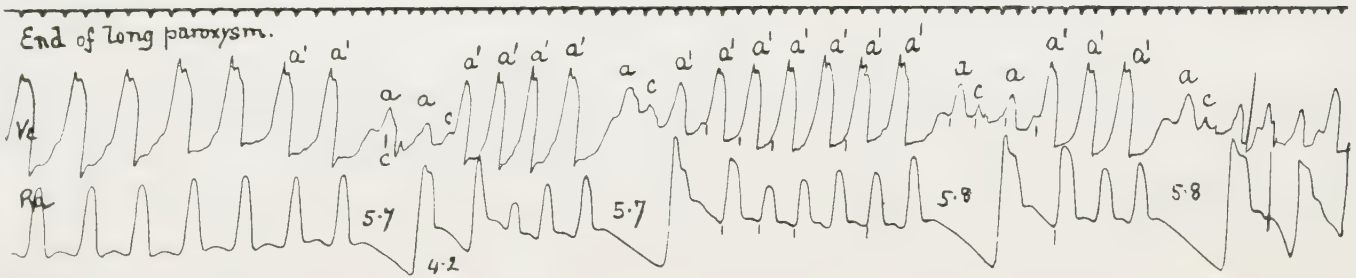


Fig. 214. (*Heart, 1910-11, II, 134.*) The end of a long paroxysm. Three shorter paroxysms of a similar nature succeed it. The paroxysmal beats were of supraventricular origin; the venous pulse during the rapid heart action was of the ventricular form, as a result of simultaneous contraction of auricle and ventricle.

These features are clearly seen in the accompanying figures (Fig. 215 and 216) in which beats from the normal rhythm and from the paroxysm are placed side by side ; curves from the three leads are shown. The constancy of the ventricular deflections, even in their smallest detail, in corresponding leads of the slow and fast rhythm, is excellently displayed in this figure ; the auricular complexes alone change ; during the paroxysms they are inverted in leads *II* and *III*.

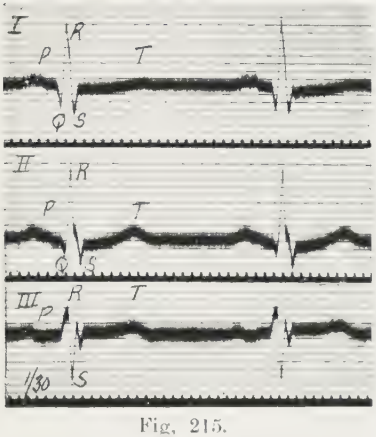


Fig. 215.

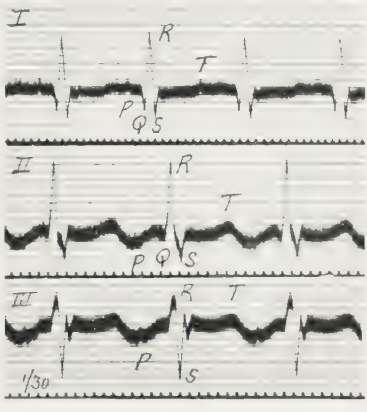


Fig. 216.

Fig. 215 and 216. Two sets of curves from a case of simple paroxysmal tachycardia. Fig. 215 was taken while the heart was beating slowly ; Fig. 216 while it was beating rapidly. The curves demonstrate the supraventricular origin of the paroxysm. The inversion of *P* in leads *II* and *III* of Fig. 216 indicates that it arose in an ectopic auricular focus. Note the precise similarity of the ventricular elements in corresponding leads of the two series. Time in thirtieths of a second.

Two other examples of auricular paroxysms are illustrated in Fig. 217 and 218. Fig. 217 was taken from a continued paroxysm in a patient who suffered from mitral stenosis ; the auricular complex is upright and falls with the preceding *T* deflection.

The similar origin of paroxysmal beats and the extrasystolic beats, interrupting the slow natural rhythm is illustrated by Fig. 218. The figure opens by showing the last five beats of a paroxysm : the post-paroxysmal pause follows and is terminated by a single normal cycle ; this in turn is succeeded by a pair of extrasystoles of auricular origin, the first ventricular response being aberrant ; two normal cycles follow. In this curve the auricular complexes of the paroxysmal stage are of smaller amplitude than those of the normal cycles and rise more steeply : the auricular complexes of paroxysm and extrasystoles are alike. It may be noted, in parenthesis, that isolated aberrant ventricular contractions, such as are shown in Fig. 218, are commonly seen in patients who suffer from

paroxysmal tachycardia. I have published another striking example of the same kind (*433*).

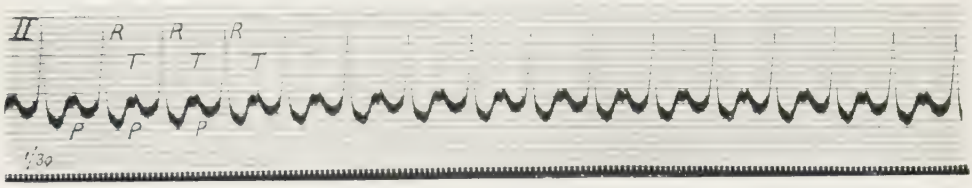


Fig. 217. Electrocardiogram from a continued paroxysm of tachycardia in a case of advanced mitral stenosis. The paroxysm arose in the auricle. Time in thirtieths of a second.

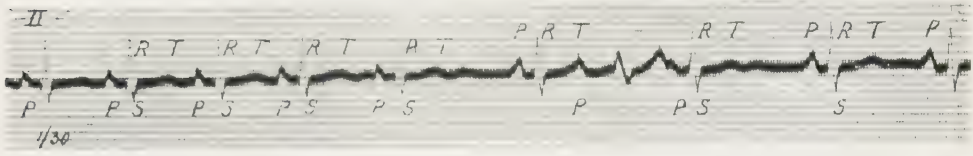


Fig. 218. The end of a paroxysm of tachycardia of auricular origin and the commencement of a slow normal rhythm, interrupted by premature contractions. These are auricular and a pair of them is shown, the former of the two yielding an aberrant ventricular response. The time-marker in this curve rules vertical lines; a pair of lines occurs at each thirtieth of a second.

A-V nodal origin.—Paroxysms in which simultaneous contraction of the auricle and ventricle occur were first published by Pan (*585*) and Rihl (*630*). Their curves were polygraphic. Of paroxysms arising in the *A-V* node and characterised by a slight reduction of the *As-Vs* interval, I have recorded two examples electrocardiographically (*434, 487*). In the venous curve, the customary *a* and *c* waves give place to conspicuous combined waves and the calculated *a-c* interval is reduced. In Fig. 219 the normal interval is 0·2 of a second, while during the paroxysm it falls to 0·06 of a second. An electrocardiogram, accompanied by a radial curve from the same patient is shown in Fig. 220. It shows the onset of a similar paroxysm, which opens with two extrasystoles, presumably of auricular origin, and continues as a regular paroxysm of beats of *A-V* nodal origin. The last beats present an inverted auricular complex and a *P-R* interval shortened from 0·14 to 0·08 of a second.

Another example is shown in Fig. 221 and 222; the lower curve illustrates the normal rhythm, the upper curve illustrates a paroxysm in which the *P-R* interval is reduced and *P* inverted. Both the normal rhythm and the paroxysm are interrupted by extrasystoles from a distinct auricular focus.

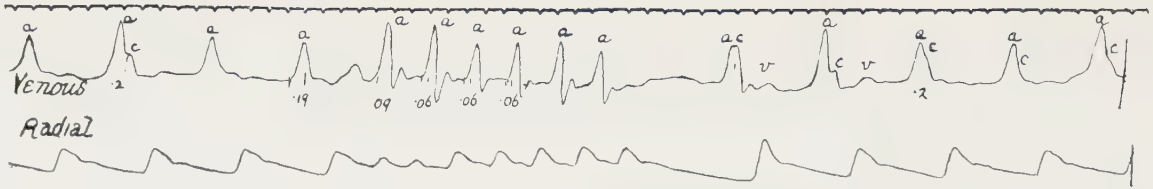


Fig. 219. (*Heart, 1909-10, I, 306, Fig. 27.*) ($\times \frac{2}{3}$.) Venous and radial curves showing a complete paroxysm arising in the *A-V* node. The *a-c* interval is reduced from the normal of 0·2 sec. to 0·06 sec. during the paroxysm. The *a* waves of the paroxysm are conspicuous.

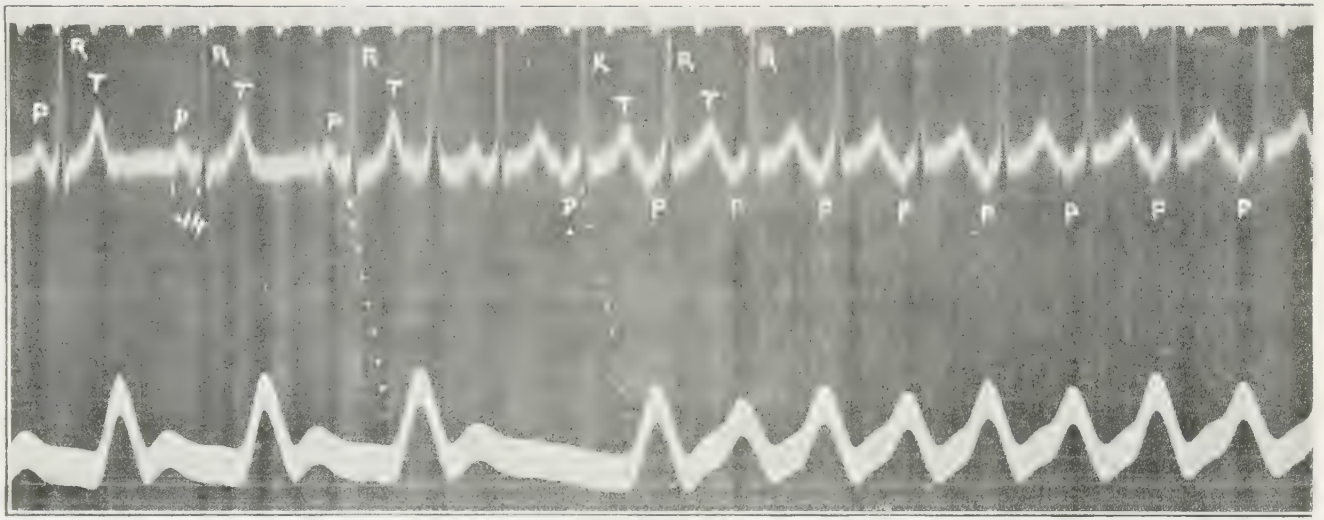


Fig. 220. (*Heart, 1909-10, I, 306, Fig. 26.*) Electrocardiogram and radial curve showing the onset of a paroxysm arising in the *A-V* node. Three normal beats are followed by two abortive contractions which appear to be of auricular origin, and the rest of the paroxysm is composed of *A-V* nodal beats, the *P-R* interval being reduced and *P* being inverted. Time in fifths of a second.

The beginning and end of a brief paroxysm of *A-V* nodal origin is shown electrocardiographically in Fig. 223. The inversion of *P* and the shortening of the *P-R* interval is excellently displayed in this curve.

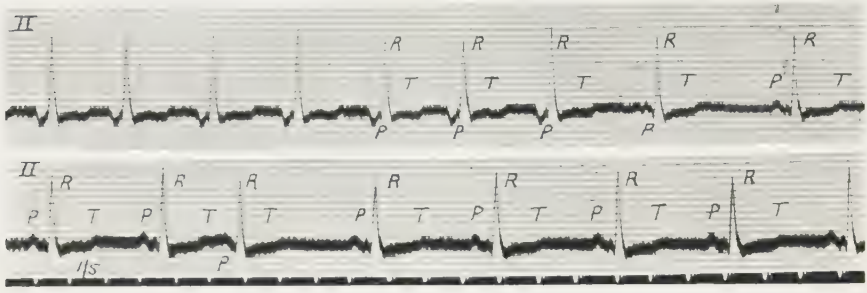


Fig. 221 and 222 ($\times \frac{9}{10}$.) Two curves from a case of simple paroxysmal tachycardia. Fig. 222 is from the period of slow and Fig. 221 is from the end of a period of rapid heart action. The curves show the auricular but ectopic origin of the paroxysm. Both slow and fast rhythms are interrupted by occasional premature beats having a common and auricular focus of origin. Time in fifths of a second.

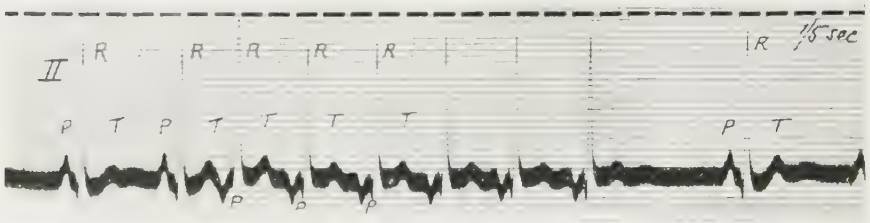


Fig. 223. ($\times \frac{9}{10}$.) A short paroxysm of six beats shown electrocardiographically to be of *A-V* nodal origin. *P* is inverted during the progress of the paroxysm and the *P-R* intervals are shortened. I am indebted for this curve to Dr. John Parkinson. Time in fifths of a second.

Paroxysms of supraventricular origin in which the ventricular form of venous pulse is seen.—In many examples of paroxysmal tachycardia the venous curve is of the ventricular form (Fig. 214); in these it is not always possible to locate the seat of disturbance, though in all we probably have to deal with simultaneous contraction of auricle and ventricle. The difficulty is that there may be no visible trace of auricular complex in the electrocardiogram (*324*, *446*, *488*). In describing the varieties of electrocardiogram accompanying the simple and slow form of *A-V* rhythm (Chapter XV), it was stated that when auricle and ventricle begin their systoles simultaneously, the abnormal auricular complex is superimposed upon the ventricular complex. Consequently, it may be impossible to trace the

auricular complex in the composite curve ; it is especially difficult when the form of the auricular complex is unknown. Most paroxysms of tachycardia presenting similar electrocardiograms to those of Fig. 225 are probably due to simultaneous contraction of auricle and ventricle. Simultaneous contraction is occasionally demonstrable, and in some instances the mechanism can be elucidated completely.

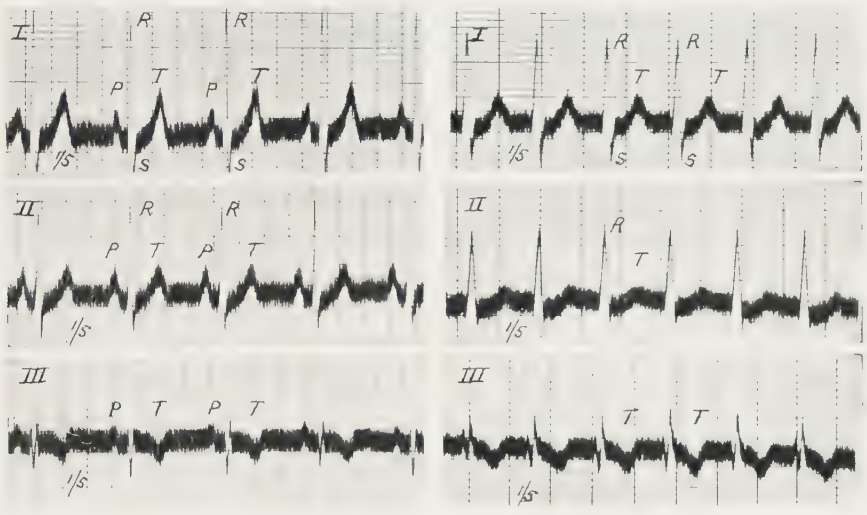


Fig. 224 and 225. ($\times \frac{9}{10}$.) Two sets of electrocardiograms from the same patient ; Fig. 224 shows the natural rhythm, the heart rate being 80 per minute ; Fig. 225 shows a paroxysm in which *P* vanishes in all leads, the rate being over 180 per minute. During the paroxysm the venous curve was of the ventricular form. Time in fifths of a second.

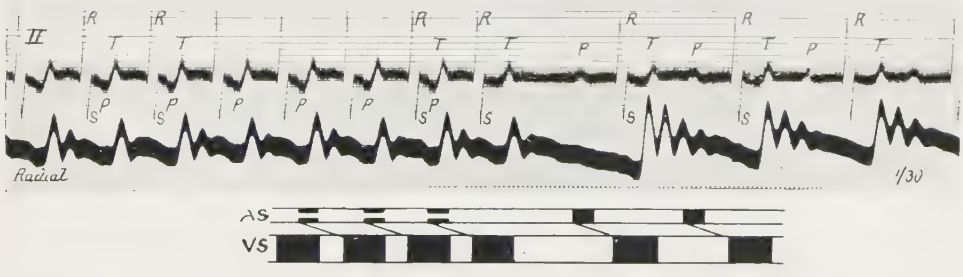


Fig. 226. ($\times \frac{4}{5}$.) The end of a simple paroxysm of tachycardia which arose in the auricle. There is delay in conduction during both slow and fast periods ; during the latter the auricular contraction falls with the preceding ventricular systole. During the paroxysm the pulse shows alternation. There is also slight alternation in the heights of *R* summits, the *larger R* corresponding to the smaller pulse beat. The venous curve was of the ventricular form and is shown in Fig. 208. Time marker in thirtieths of a second.

The end of a paroxysm is shown in Fig. 226. If the ventricular complexes of the slow and fast periods are compared, they are found to be alike, except that the upstroke of T is preceded by a dip during the stage of the paroxysm. This difference is not incompatible with change of rate alone; but such a cause can be excluded in the present example. The change in T is due to the superimposition of an invert P upon it while the heart is beating rapidly. Comparison of the last two cycles elicits this fact, for the last T deflection is uncomplicated by an auricular systole. The absence of a P deflection at the end of a paroxysm also unmasks the sequence of chamber contraction; evidently the ventricle is responding to the auricle, though the P-R interval is increased. Had the last T of the paroxysm also been complicated by an invert P the curves would have declared the paroxysm to be of A-V nodal origin. In this curve the natural P-R interval is prolonged, as the end of the same figure clearly demonstrates, at the resumption of the S-A rhythm. The venous curve of this patient is shown in Fig. 208, page 243. It seems therefore that paroxysms of the supraventricular type, which are accompanied by the ventricular form of venous pulse and in which the auricular complex is lost or not clearly distinguished, may result in one of two ways :—(*a*) when the paroxysm arises in the A-V node, and (*b*) when contraction is simultaneous in auricle and ventricle because the conduction interval is increased (*4, 446*).

A paroxysm of A-V nodal origin, in which the auricular complexes are clear though buried in the ventricular complexes, is shown in Fig. 228. This paroxysm arose from a relatively low region of the A-V node, the ventricle contracting somewhat before the auricle. There is a Vs-As interval.

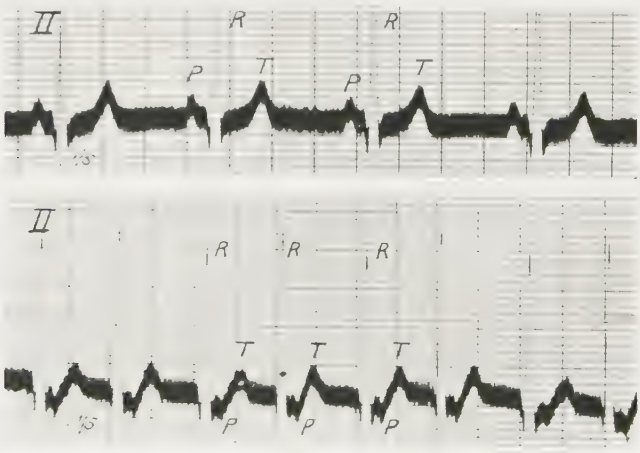


Fig. 227 and 228. Electrocardiograms from a patient after and during a paroxysm of tachycardia arising at a low level of the A-V node. During the paroxysm (Fig. 228) the auricular complex is inverted, and falls after the commencement and during the inscription of the ventricular complex. Time in fifths of a second.

Paroxysms of supraventricular origin are characterised in electrocardiograms by the shape of the ventricular complexes; the natural type of ventricular curve is maintained in all, with the solitary exception of rare paroxysms to be described in the succeeding chapter. To further locate the point from which the tachycardia springs, the positions of the auricular systoles must be ascertained, and should be studied especially at the onset and ending of the paroxysm (*446*). In all instances fully studied, the auricular representations have been anomalous, indicating ectopic impulses. The origin may be in an abnormal auricular focus, it may be in the *A-V* node. Paroxysms arising in the *S-A* node have not as yet been identified.

Chapter XXI.

Paroxysmal Tachycardia in which the Ventricular Systoles are Abnormal.

If a dog's ventricle is stimulated rhythmically by electric shocks of sufficient intensity, the ventricle responds to each stimulus and, as stated in a former chapter, the auricle after a while follows suit. The beat of the whole heart is then controlled by the stimuli but its action is reversed; despite this reversal and the mechanical disadvantages under which the heart labours, the circulation may be sustained efficiently for considerable periods of time. The number of ventricular cycles which may pass before the auricle yields its first response is variable. The beats may be retrograde after one or more cycles (Fig. 171, page 216, and Fig. 196, page 234); on the other hand, the faster rhythm excited in the ventricle and the slower rhythm propagated in the auricle from the *S-A* node, may remain independent for a short or long period (Fig. 236). When the beats are retrograde, the two chambers beat simultaneously, the ventricle preceding the auricle by an interval equal to or in excess of the natural *As-Vs* interval. Where this mechanism prevails, the ventricular form of venous pulse is to be anticipated and is proved to occur in experiment (Fig. 235). In this figure the *c* and *a* waves are distinct even during the stage of rapid heart action, the former leading by a considerable interval; in other crude polygraph curves the two waves may be fused and inseparable.

In electrocardiograms the ventricular complexes of the excited beats are abnormal, and their configuration is governed by the region of the ventricle to which the stimuli are applied (see Chapter XVII). Falling with each ventricular complex is an anomalous auricular complex (Fig. 196, page 234): it is anomalous because the course of the contraction wave in the auricle is abnormal. As might be expected, the form of this anomalous complex, simulates that of auricular contractions of *A-V* nodal origin; it is either inverted or comprises several phases (usually a downward followed by an upward movement).* Being buried in the abnormal ventricular complex it is not always easy to decipher in its detail. The form of these auricular complexes (for examples of which see Figs. 196—199, pages 234 and 235, and

* As first recorded by Hering (*282*) and myself (*432, 447*).

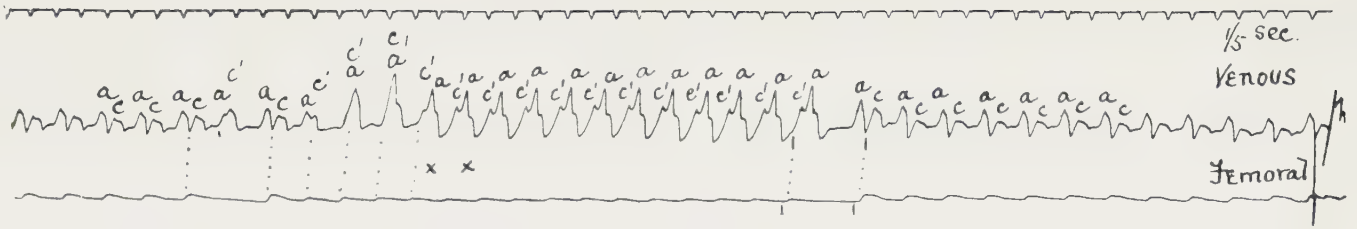


Fig. 235. (*Heart, 1910-11, II, 141, Fig. 8.*) A polygraphic curve, from a dog, showing the form of venous and femoral curves during a tachycardia of ventricular origin (excited by successive induction shocks). To the left the normal rhythm is seen, each beat is accompanied by *a* and *c* waves. The rhythm is first interrupted by a single premature beat *c'*, one normal cycle follows, and then the ventricular tachycardia commences. It becomes retrograde at the fifth beat and from this point onwards the venous pulse cycles are uniform in appearance, and the " ventricular form of venous pulse " is present.

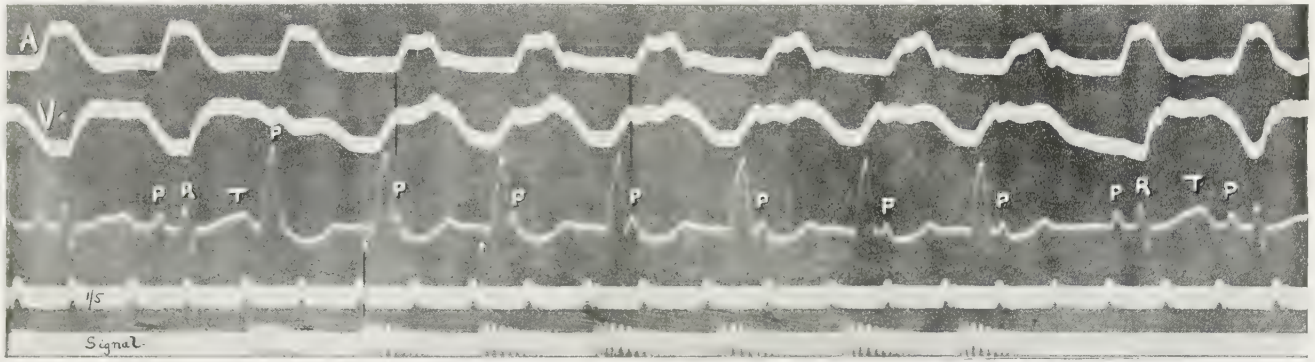


Fig. 236. Auricular and ventricular myocardiograms and electrocardiograms (lead *II*) from a dog. The right ventricle has been stimulated with seven rhythmic electric shocks (see signal); the corresponding responses of the ventricle are seen. Throughout, the auricular rhythm remains undisturbed; witness the auricular muscle curve *A* and the regularly placed summits *P*, which fall with the ventricular complexes during the accelerated heart action.

Fig. 241), and of the similar complexes in *A-V* rhythm, has been much discussed. A summary will be found in Kahn's monograph (*367*).

When the heart is beating in retrograde fashion and either the ventricular action is very rapid or the *A-V* bundle is conducting inefficiently, the auricles may fail to respond (*432*) and a condition of reversed heart-block then manifests itself (Fig. 238); the degrees of reversed block are similar to the degrees of forward *A-V* heart-block.

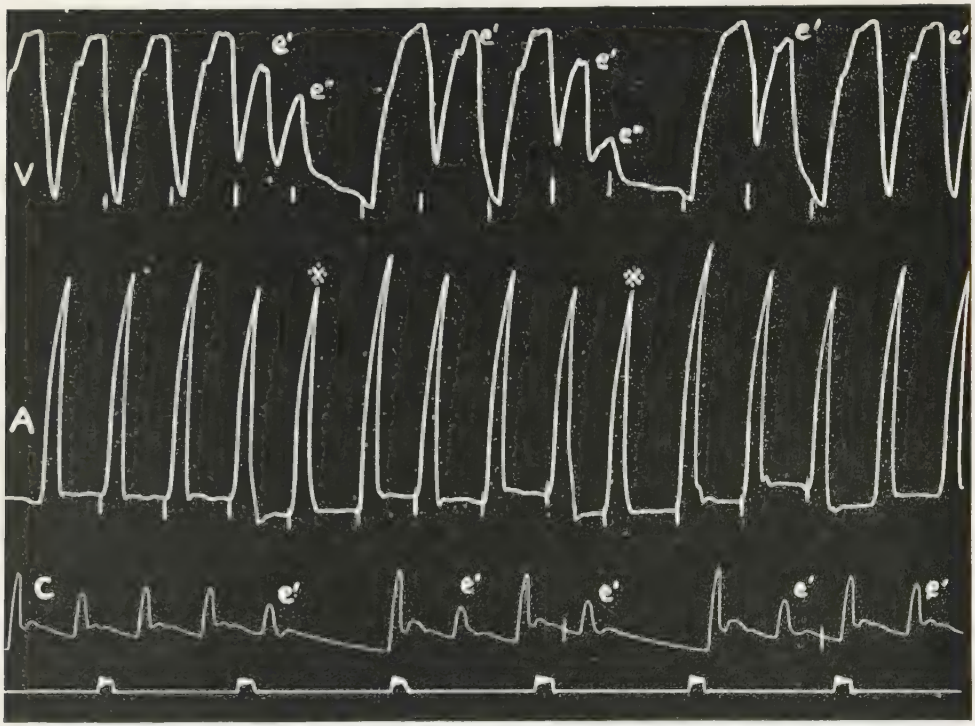


Fig. 237. (*Heart, 1909-10, I, 104, Fig. 1.*) Myocardiographic curves (*V* = ventricle, *A* = auricle) and Hürthle carotid pressure curve (*C*). A curve taken from a dog shortly after ligation of the right coronary artery, and showing premature ventricular contractions, single (*e'*) and successive (*e'*, *e''*). In two instances a pair of premature contractions occurs, the second awakens an auricular response on each occasion; the premature auricular beats are marked with asterisks. An instance of the mechanism which precedes tachycardia of ventricular origin. Time in seconds.

Clinical examples.

As in the case of auricular, so in the case of ventricular extrasystoles, the premature beats may not be isolated but may occur in pairs. Pairs of ventricular extrasystoles (Fig. 239) are not very uncommon, groups of them or short paroxysms are less frequent.

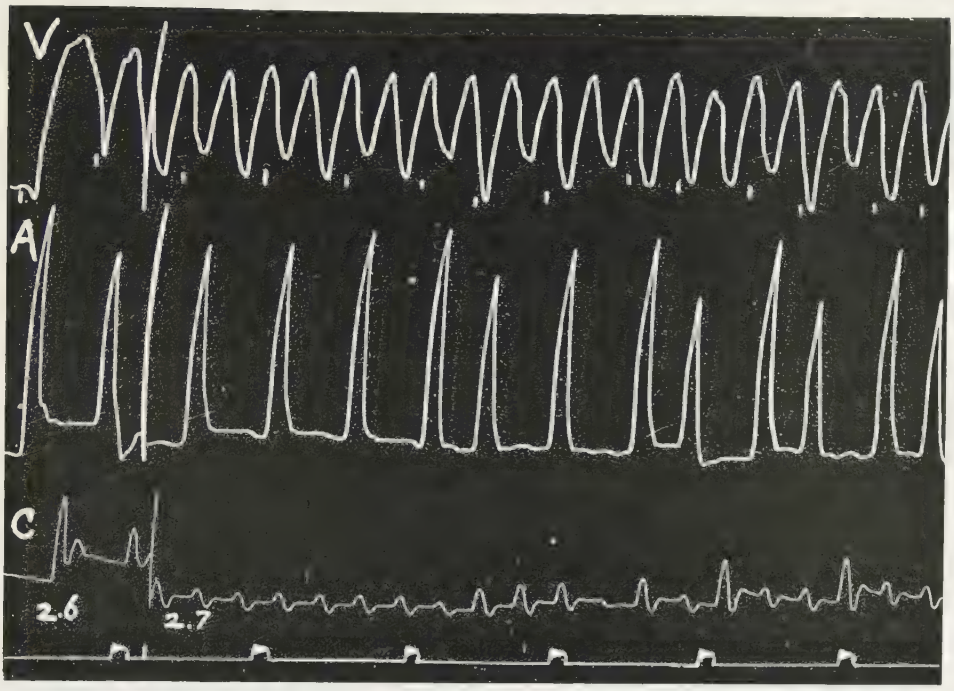


Fig. 238. (*Heart, 1909-10, I, 112, Fig. 7.*) Myocardiographic curves (*V* = ventricle, *A* = auricle) and Hürthle carotid pressure curve (*C*). To the left of the index marks one normal and one premature ventricular contraction are shown. To the right (one minute later) the ventricle is in tachycardia and the auricle is responding to each second or to two in three ventricular beats (reversed heart-block). The ventricular rate is approximately 220. The disturbances were the result of obstructing the right coronary artery. From a dog; time in seconds.

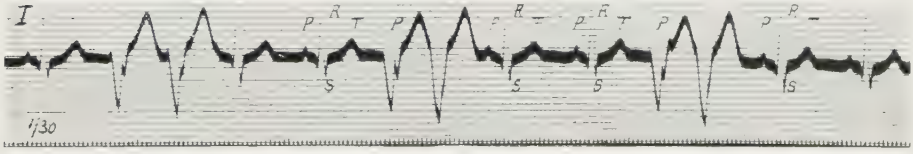


Fig. 239. (× $\frac{4}{5}$.) Electrocardiogram from a patient showing three pairs of extrasystoles arising in the ventricle. Time in thirtieths of a second.

I have published (*447, Fig. 132*) an example in which a paroxysm of six beats of ventricular origin interrupts the normal rhythm; it is seen in Fig. 240; later the curve shows a solitary interruption arising in the same focus. Measuring the distance between the clearly defined *P* deflections in this curve, the positions of the buried auricular summits may be estimated,

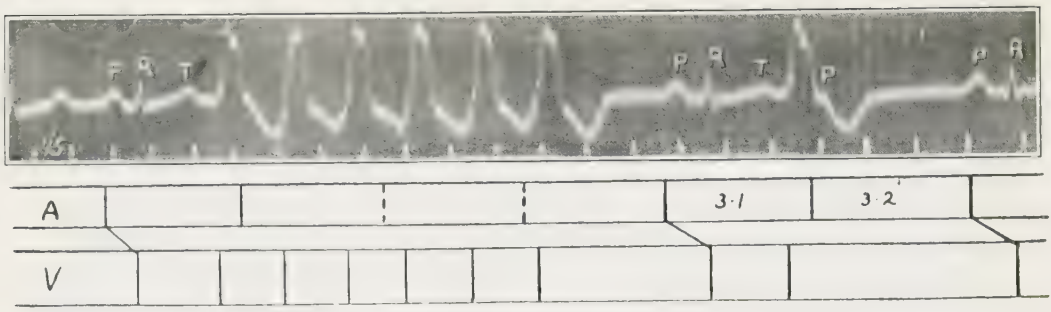


Fig. 240. An electrocardiogram from a patient, showing a paroxysm of six beats originating in the right ventricle and a solitary beat of the same kind. The auricular rhythm appears to be undisturbed throughout (see diagram below). The time is in fifths of a second; it should be noted that the photographic paper was not travelling at a quite uniform rate.

for in this example of a ventricular paroxysm the beats do not appear to have been retrograde and the auricular rhythm seems to have been undisturbed. Similar examples have been recorded by Hunt (*45*), by Cohn (*63*), and by Vaughan (*738*). Hart (*240*) has published very beautiful clinical curves of paroxysms lasting from a few seconds to several minutes. One of his curves, which shows a short paroxysm, is reproduced in Fig. 241. The curve

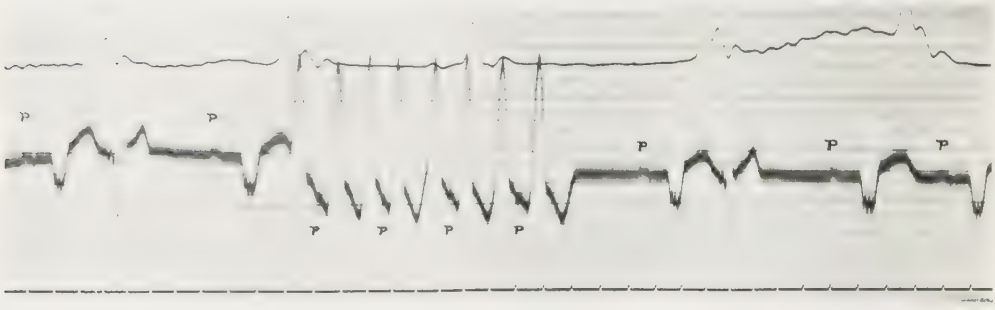


Fig. 241. (*Hart. Heart, 1912-13, IV, 128, Fig. 8.*) A paroxysm of tachycardia of ventricular origin. There are eight beats in the paroxysm and an invert auricular complex is seen on the alternate downstrokes of the ventricular complexes. The paroxysm is retrograde but alternate impulses alone awaken auricular responses. Time in fifths of a second.

presents a paroxysm of eight beats, arising in the right ventricle. Alternate impulses are retrograde to the auricle and the corresponding anomalous auricular deflections are seen where Dr. Hart has marked them on the alternate downstrokes; alternate impulses are blocked.

Now these examples are clear instances of paroxysms arising in the ventricle; they are unmistakable because the first beat of the paroxysm

has the same relations to the preceding normal rhythm as has a ventricular extrasystole. When records are obtained during the progress of long paroxysms the analysis is far less certain. In Fig. 242 a set of curves is reproduced from a patient who exhibited tachycardial periods having an abrupt onset and ending; side by side with these is a second series from the same leads (Fig. 243) showing the mechanism two days later and after the paroxysm had subsided. The paroxysm would seem at the first blush to be of ventricular origin, for the ventricular complexes are anomalous and the notch towards the end of the complex in leads *II* and *III* probably represents an abnormal auricular systole. Yet this origin is not certain, for an alternative interpretation is equally plausible, namely, that the paroxysm is in reality auricular; the view being that the ventricle is responding to the preceding auricular impulse after an increased conduction interval, and that the excitation wave takes an aberrant course in the ventricle: aberration is known to be a frequent phenomenon in patients who are the subjects of paroxysmal tachycardia. It is impossible to decide the exact origin of a paroxysm of the kind illustrated in the present figure unless its first or last beat is recorded.

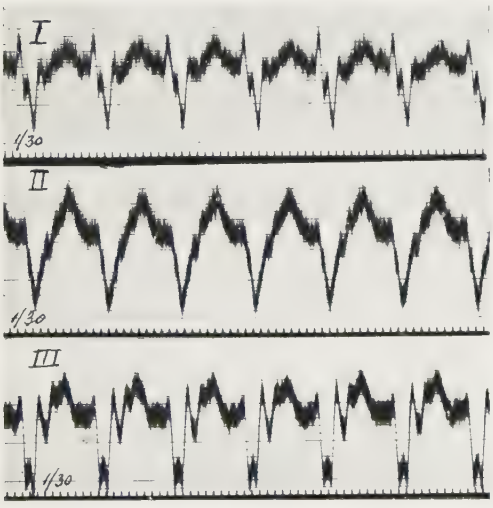


Fig. 242.

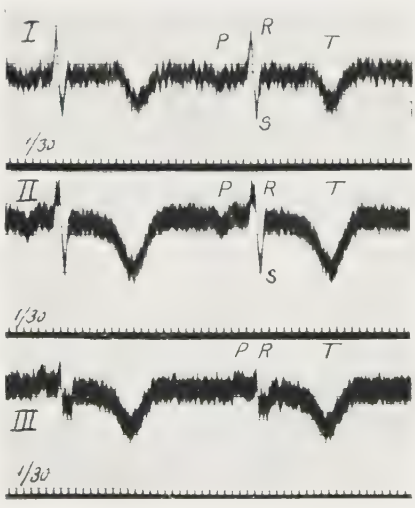


Fig. 243.

Fig. 242 A series of curves from the three leads during a paroxysm of tachycardia of indeterminate origin.

Fig. 243. The corresponding series after the resumption of the natural rhythm. Time in thirtieths of a second.

An example of an auricular paroxysm, simulating a ventricular paroxysm, is shown in the next figures. Fig. 244, 245 and 246, were taken from a patient who exhibited frequent extrasystoles of auricular origin and short paroxysms of tachycardia. In Fig. 244 a single auricular extrasystole is

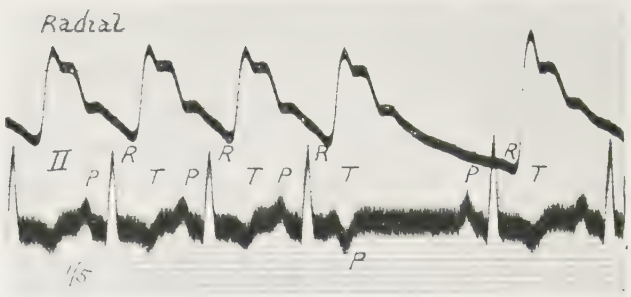


Fig 244. Radial and electrocardiographic curve; a single extrasystole arises in the auricle and deforms the third *T* deflection; its impulse fails to reach the ventricle. Time in fifths of a second.

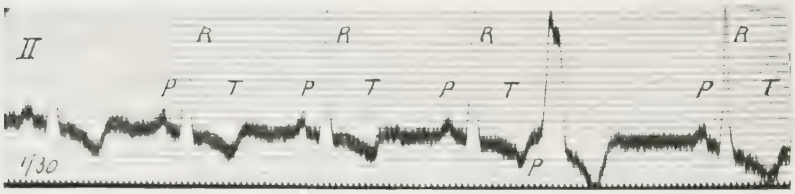


Fig. 245. Electrocardiogram from the same patient and showing a similar auricular extrasystole; the excitation wave of the responding ventricular systole pursues an aberrant course. Time in thirtieths of a second.

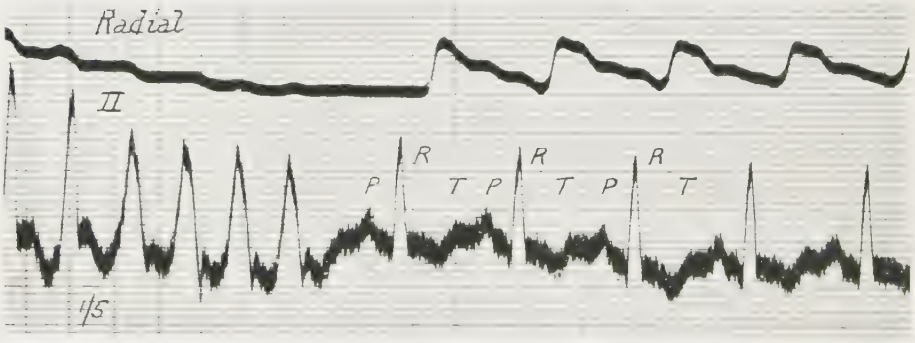
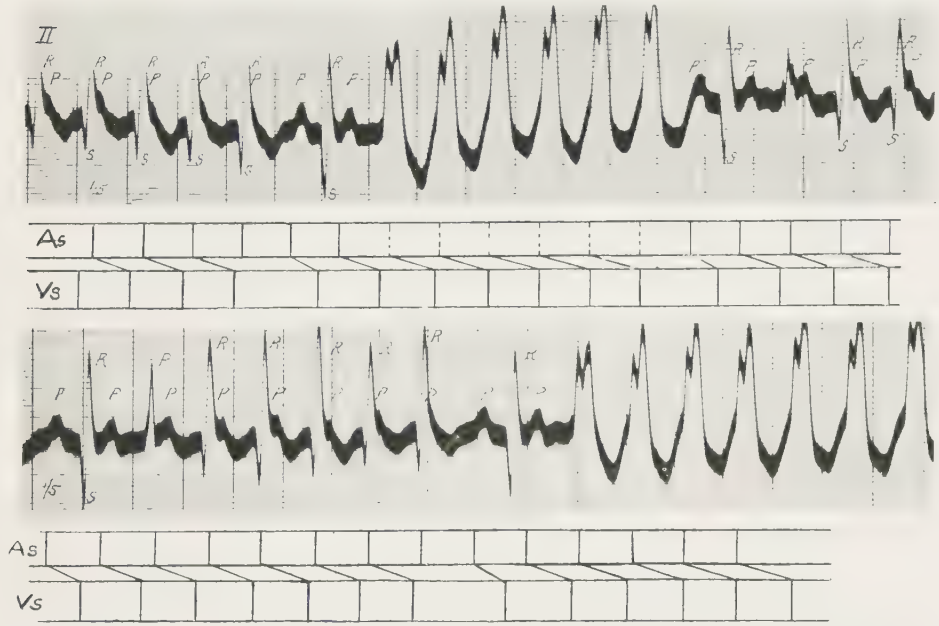


Fig. 246. Radial and electrocardiographic curves from the same patient, showing the termination of a paroxysm of tachycardia. Comparing the abnormal beats with that of Fig. 245, it is probable that the paroxysm arose in the auricle. Time in fifths of a second.

shown, but it yields no ventricular response ; the curve displays a conduction defect in the *A-V* system. In Fig. 245 a similar extrasystole is followed by an anomalous ventricular complex due to conduction of the impulse to the right ventricle only ; the curve displays a conduction defect in the left division of the *A-V* bundle. Fig. 246 shows the termination of a paroxysm of beats of the same type. Here there is strong presumptive evidence that the paroxysm arose in the auricle, and that the left bundle division failed to conduct while the heart's action was rapid. A still more convincing example of aberration during a continued paroxysm of accelerated heart action is provided by Figs. 247 and 248.



Figs. 247 and 248. ($\times \frac{3}{4}$.) Two electrocardiograms from a child, and corresponding explanatory diagrams. The auricle is beating at 290 per minute and the ventricle is responding : the *P-R* intervals are increased and occasionally a ventricular response is missed. From time to time also, certain of the Purkinje tracts fail to distribute the excitation process normally to the ventricle and the ventricular complexes become anomalous. Time in fifths of a second.

These curves were taken from a child in whom the auricular rate approximated 290 per minute. Examining the lower curve first of all, we see a series of seven ventricular beats, each a response to a preceding auricular impulse (*P*) ; but the *P-R* interval is gradually increasing as the cycles succeed each other, up to the point where the 8th auricular impulse fails to yield a response. A single ventricular contraction is missed ; response to the

9th auricular impulse and to the 10th occurs, but this last-named impulse is followed by an aberrant ventricular complex, the first of a whole series. In the upper curve similar events are shown, but here the series of aberrant beats is fleeting; when the customary form is resumed, the auricular complexes once more become recognisable. The point to be emphasised is that the last auricular summits fall exactly into place to continue in series with the first auricular summits of the curve. There has been no disturbance of the auricular rhythm. The series is therefore drawn as complete in the explanatory diagram. The anomalous ventricular beats in both curves hold the precise time relations to the auricular contractions, recognisable or calculated, which are to be anticipated if all the beats of the ventricle are responses to the auricle.*

Thus it appears that in the human subject paroxysms presenting anomalous ventricular complexes may be produced in one of two ways; these paroxysms either arise in the ventricle itself, or, arising in the auricle, the excitation wave pursues an abnormal ventricular course.

* Almost identical curves have been published by White and Stevens (*770*).

Chapter XXII.

AURICULAR FLUTTER.

In the present chapter a condition is considered which in many respects resembles the paroxysms of tachycardia of auricular origin discussed in Chapter XX. Yet in its fully developed form it differs to such an extent from these paroxysms that for the present it has to be considered as a separate affection. It is, however, to be acknowledged that the dividing line between the two mechanisms, the simple paroxysmal and the attack of flutter, cannot be drawn rigidly and that at a future date their separation will prove to be unnecessary if it is shown that the differences between them are purely differences of degree. The facts remain that when a new rhythm arises in the human auricle and its rate approaches and surpasses 300 per minute, the heart chambers respond in a curious fashion, and that certain qualities and associations of this new rhythm seem to be almost peculiar to it. We may divide the simple paroxysm from the attack of flutter arbitrarily by defining the latter as a new rhythm whose rate surpasses 200 and may reach 350 per minute. There is a group of patients in whom the auricles beat constantly or transiently at rates of about 300 per minute; this is the group which supplies us with our present data.

Experimental flutter.

When the mammalian auricle is stimulated by successive induction shocks and is responding to each shock, the rapid heart action ends immediately the stimuli cease. But when the stimuli follow each other so rapidly that the auricle is only just capable of responding to each stimulus, or when the auricle is excited by means of a weak faradic current, the accelerated and regular action may continue for a little while or for considerable periods of time after the stimulation ceases (Fig. 249).* It seems clear that a relation exists between the rate of the auricular responses and the tendency for the accelerated action to persist, but it must be confessed that the precise conditions

* The end curves of fibrillation which Rothberger and Winterberg (*674*) describe in detail, and to which they have applied the term flutter, are for the most part not identical with clinical flutter. I say so because the action of the auricle in their curves is not sufficiently regular. This matter is more fully discussed on page 328.

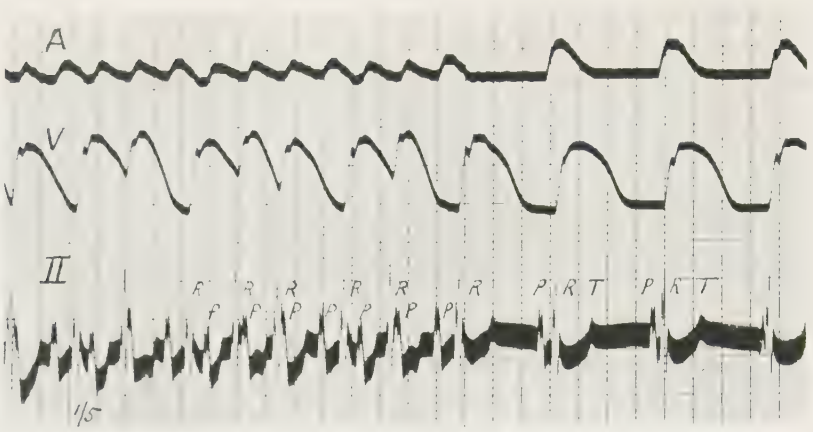


Fig. 249. A curve taken from a dog and illustrating auricular flutter. The auricle was stimulated by means of a weak faradic current and a greatly accelerated heart action was thereby induced. The rapid action continuing after the end of stimulation is shown. It soon terminates and the natural action of the heart is then resumed. During the period of tachycardia the auricle beats more rapidly than the ventricle; a condition of partial heart-block is present. A = auricular, and V = ventricular myocardiogram. Time in fifths of a second.

which conduce to persistence are still obscure, and that it cannot be brought about at will by stimulation or other known means. Occasionally an extremely rapid and persistent auricular beating appears spontaneously during the course of an experiment and remains unexplained. In a few animals I have been able to induce repeated exacerbations of auricular rate by injecting glyoxyllic acid into the blood-stream; the disorder resembled flutter (Fig. 250), but, as the auricular waves were not precisely regular, cannot be declared positively to be of this kind. If spontaneous flutter appears in animals it may persist for a long while or may stop shortly; when it stops it does so without warning, and after a pause the natural rhythm is resumed. In this respect it resembles clinical paroxysms of simple tachycardia. It differs from these in that the rate is so great that the ventricle usually fails to respond to more than one impulse in two.

The term flutter was first used by McWilliam (*522*), in describing the response of the auricle to faradic stimulation. It has been adopted by Jolly and Ritchie (*355, 642, 645*) and, since the publication of their paper, by other writers to designate a special condition in patients.*

Clinical flutter.

In man, acceleration of the auricle to a rate of 300 beats per minute or thereabouts is not very uncommon (*457*); generally speaking, it is an

* The nature of the response which McWilliam observed cannot positively be identified with flutter or fibrillation at the present time, but it was probably coarse fibrillation.

established condition, thereby differing from simple paroxysms, though in some patients the attacks are transient and repeated (*515, 3rd ed.*). According to Hertz and Goodhart (*304*), who first drew attention to the condition in man,* the auricular movements may be visible upon a

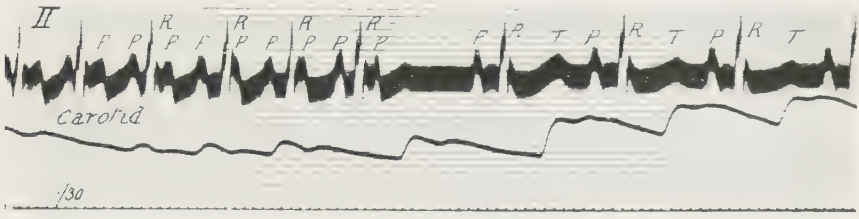


Fig. 250. ($\times \frac{9}{10}$.) The end of a period of auricular disorder, produced in a dog by injecting glyoxyllic acid into the blood-stream. In the first portion of the curve the auricular waves follow each other at 492 per minute, the ventricle is beating at a rate of approximately 246 per minute. Time in thirtieths of a second.

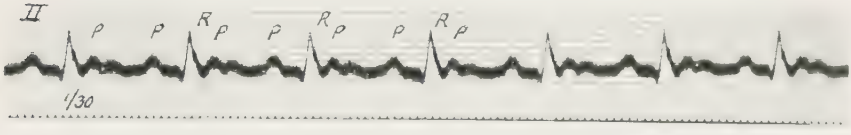


Fig. 251. ($\times \frac{9}{10}$) A curve from a patient, for comparison with Fig. 250. It shows an auricular rate of 226 per minute, and a ventricular rate of 113 per minute. Time in thirtieths of a second.

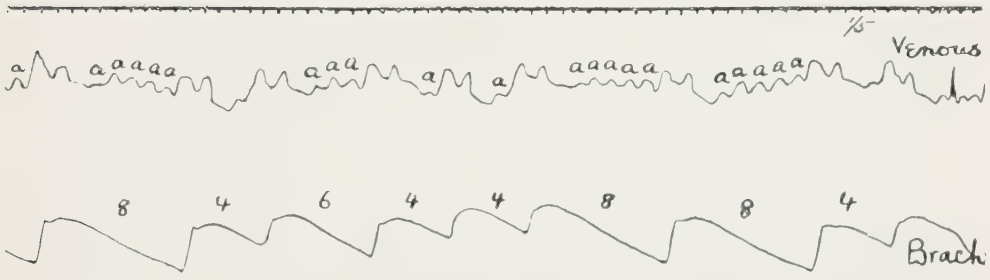


Fig. 252. (*Heart, 1912-13, IV, 201, Fig. 18.*) Venous and arterial curve in a case of auricular flutter. The regular and small auricular waves are distinct in the jugular curve, especially in the long diastoles. The arterial pulse is irregular, the numbers set against the pulse beats represent the numbers of auricular beats corresponding to individual ventricular cycles.

fluorescent screen; they may be audible with the stethoscope, though uncountable because of their rapidity. They are to be recorded from the

* Ritchie, in 1905, published a record (*641*) of the auricles raising their rate to 275 per minute after an injection of atropine. The record is almost certainly one of flutter, though induced in this manner.

jugular veins (*304*) in many patients, especially in those in whom the ventricular rate is relatively slow and the diastoles consequently long (Fig. 252); but when the ventricular action is rapid, and this is the rule rather than the exception, it is usually impossible to disentangle them from the ventricular

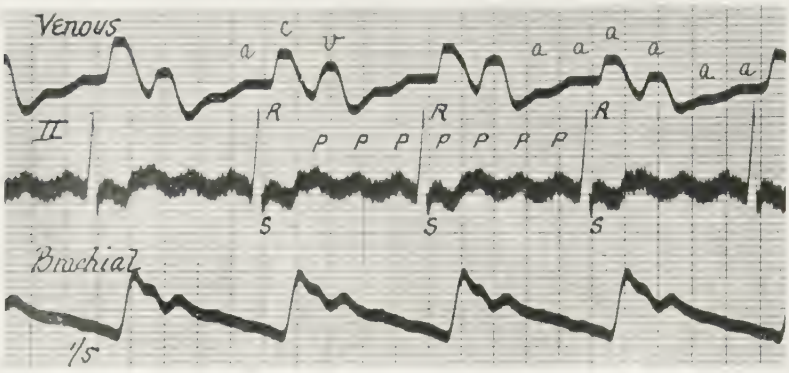


Fig. 253. Venous, electrocardiographic and brachial curves from a case of auricular flutter in which the auricle beat four times as fast as the ventricle. The venous curve superficially resembles a normal curve, and is apt to be interpreted as consisting of three chief waves to each cycle, namely, *a*, *c* and *v* (as marked to the left).

The true interpretation of the events is shown to the right; all the venous waves are auricular in origin, those which fall in the period of ventricular systole being prominent, while those which fall in diastole are insignificant. Time in fifths of a second.

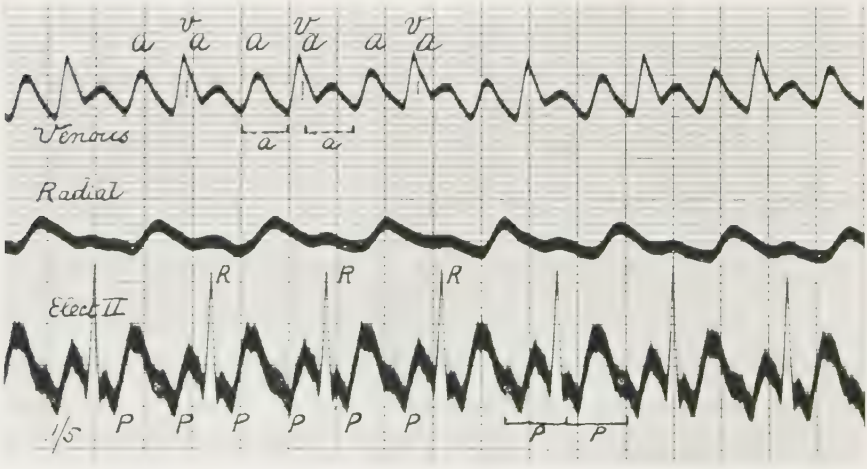


Fig. 254 Electrocardiogram, venous and radial curves from a case of flutter. The auricle is beating at 250 per minute, the ventricles at 125. In the jugular curve one wave *a* is distinct; the second falls and fuses with *v*; most of the force of this auricular contraction is lost, for it comes at a time when the auriculo-ventricular valves are open. Time in fifths of a second

jugular waves which are themselves close set. Even when the ventricular rate is slow, many or most of the auricular waves may be too feeble to distinguish. The waves which fall with ventricular systole and the last waves in the diastole are the most distinct (Fig. 252); sometimes the venous curve

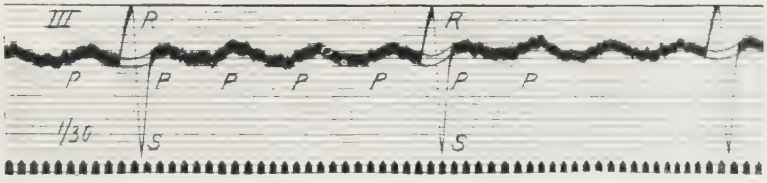


Fig. 255. (*Heart, 1911-12; III, 279, Fig. 19*) From a case of flutter, showing 4 : 1 heart-block. Those portions of the auricular complexes which have been obscured by the initial phases of the ventricular complexes have been reconstructed so as to illustrate the continuity of the auricular oscillations. The standard for this curve is 1½ centimetres to the millivolt. Time in thirtieths of a second

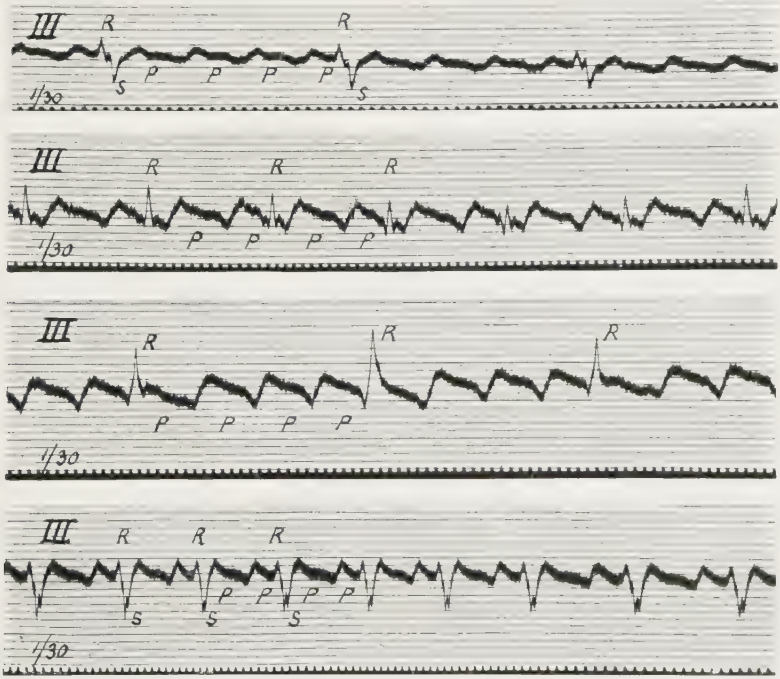


Fig. 256. Curves from lead *III* in four separate cases of auricular flutter. In the second and fourth curves 2 : 1 block is present; in the first curve 4 : 1 block is shown; and in the third curve the responses of the ventricle are irregular. Notice the resemblance of the auricular portions of the four curves. Time in thirtieths of a second.

closely simulates a normal jugular tracing (Fig. 253). Generally speaking, in electrocardiograms the auricular complexes are at once recognisable.

The auricular beats in electrocardiograms.—From patient to patient the form of the auricular complex is remarkably constant. In Fig. 256 are records from four separate patients ; in these the form is almost identical ; in many other patients of my own the curves have had the same outlines. The form in curves published by other observers is similar.*

The constancy in form from cycle to cycle in the same patient suggests the origin of the impulses in a constant focus ; so also does the regularity of the rhythm, which deserves stress.† The auricular complexes are blunt at their turning points, but if comparator measurements are taken from sharp ventricular deflections, when the ventricle is responding regularly to the auricle (*i.e.*, to each second impulse), variations in the length of cycles can be shown to be usually minute. In different cases the average variation in the lengths of intra-auricular cycles varies from 0·0009 to 0·0077 of a second. The regularity from cycle to cycle in form, in amplitude and in length, I regard as a remarkable feature of the condition.‡

The auricular complexes are contiguous when the rate exceeds 260 per minute ; the string is in constant movement, the isoelectric condition is not maintained for any measurable period ;§ in leads *II* and *III* each complex seems to begin at the upstroke ;‖ it reaches a blunt summit and descends less abruptly. The gentle downsweep is often notched. In lead *I*, the complex may scarcely be perceptible or may consist of a short and sharp summit (Fig. 257). Exceptionally and when the rate is slower the form is different (Fig. 251). It is probable, though not certain, that these

* This constancy of type from case to case suggests that there may be a similar constancy in the route travelled by the excitation wave in the auricle. Possibly it is conditioned simply by excessive rate, though this explanation is not the most plausible ; an alternative explanation, namely, that of circus movement, is discussed in Chapter XXVIII.

† The alternative explanation, given in Chapter XXVIII, is equally compatible with these facts.

‡ A very slight change in rate during deep breathing has been observed by Levine and Frothingham (*420*).

§ As the string is in constant movement it is to be assumed that some portion of the auricle is always active ; some portion of the auricle is always in systole (in the electrical sense). We must conclude, therefore, that a real diastole is lacking ; though there must be a period of rest for individual muscle elements. It is probable that at all parts of the auricular cycle, *some* portion of the auricle is active and that there is no rest in the muscle of this chamber considered as a whole. The only rest which is obtained by the first part of the auricular muscle to become active is obtained during the short interval between the subsidence of activity in this part, and the subsidence of activity in parts of the auricle to which the excitation wave has far to travel. In the normally beating heart this interval is but a few hundredths of a second ; but it is probably greater when the auricular rate is extreme, as conduction is then slower.

‖ A statement based on the form of the curve in the three leads (*457*).

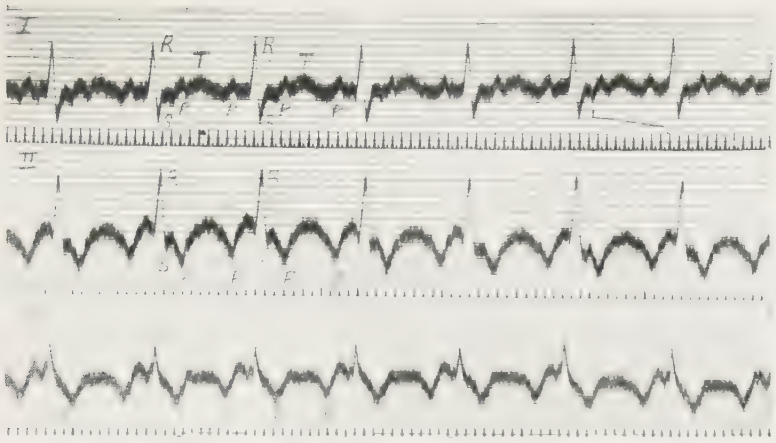


Fig. 257. (*Heart, 1911-12, III, 279, Fig. 24*). Curves from the three leads in a case of auricular flutter. The auricular rate is 324, the ventricular 162 per minute. Time in thirtieths of a second.

new rhythms are ectopic in origin ;* for the auricular complex does not resemble the normal complex in the same patient (Fig. 305, page 327).

The rate of the auricular rhythm from minute to minute, from week to week, and even from year to year when long continued, is wonderfully uniform in any given patient (*454*). Thus in 30 records taken from one patient, at intervals over a period of six months, the auricular rate always lay between 260 and 278 per minute. None of those influences, which produce retardation or acceleration of the normal rhythm, seem potent while the auricles are fluttering.† Exercise, posture (*451, 454*), effective pressure on one or other

* Uncertainty is due to the change which complexes are known to undergo when the heart rate is very greatly accelerated, although the point from which the impulses start remains constant. Of hypotheses which have occurred to me in attempting to explain flutter of the auricle, the origin of the new rhythm in the *S-A* node is one. The only argument against this hypothesis, so far as I am aware, is the frequent dissimilarity of the complexes while the heart is beating slowly and rapidly. There are several arguments in favour of the hypothesis, but none of these can be regarded as conclusive. They are :—

(1.) The tissue of the *S-A* node is special tissue and on the analogy of the *A-V* node, new rhythms might be expected to arise from it. Yet none have been recorded, unless indeed flutter has this origin.

(2.) When the auricular rate is less extreme and the auricular complexes are not contiguous (as in Fig. 249 and 251) the normal auricular complex is simulated.

(3.) When the end of a paroxysm is recorded, the last paroxysmal cycle (returning cycle) is equal in length or shorter (Fig. 249) than cycles of the normal rhythm. In this respect there is a resemblance to the sinus extrasystole (see page 229).

(4.) If flutter has its origin in the *S-A* node, the similarity of the auricular complexes from patient to patient would be explained.

(See further discussion of the cause of flutter in Chapter XXVIII.)

† See, however, the third footnote on the preceding page.

vagus (*451, 457, 635*) (Fig. 258), the administration of cardiac drugs, each and all appear powerless ; the rate is unperturbed in all these circumstances. In this respect, flutter resembles the simple paroxysm, though in flutter the readings are even more uniform.

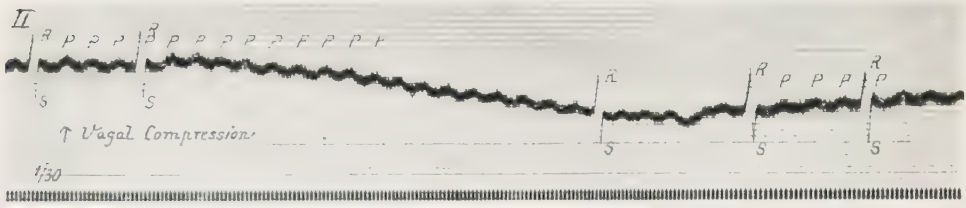


Fig. 258. (*Heart, 1911-12, III, 279 Fig. 18*). (× $\frac{4}{5}$.) The effect of pressing upon the right vagus nerve in a patient who demonstrated flutter and while the ventricle was responding to each fourth auricular impulse. The auricular rhythm remains unaltered, the ventricular rate is notably retarded. Time in thirtieths of a second.

Responses of the ventricle.—In a child (Fig. 248, page 261), the ventricle has been found to respond to almost every auricular impulse.* Usually the picture is that of 2 : 1 heart-block, maintained for long periods of time ; but occasionally the ventricular rate rises in these patients to the full auricular rate, a phenomenon generally (*515, 3rd ed.*), but not always (*34*), associated with temporary loss of consciousness. On the other hand, the responses may be less frequent, or may become so as a consequence of deficient conduction. A 4 : 1 response is not unusual, neither is a mingled 2 : 1, 4 : 1 response, nor more complex ratios (Fig. 252). Complete heart-block has been recorded and then the rate has been very slow (*355*). Stimulation of the vagus (*451, 457, 635, 644*), by decreasing conduction, always slows the ventricle (Fig. 258), though the auricular rhythm remains unaffected ; this effect is obtained in equal degree by both nerves. Digitalis and the allied drugs act in a similar manner ; given in sufficient doses they can be relied upon to produce heart-block or to accentuate a pre-existing block. Exercise or excitement exert the reverse effect. The almost constant association of auricular flutter with some grade of heart-block, and the ease with which higher grades of block may be induced, is attributable almost solely to the extreme acceleration of the auricular rhythm. The rate of the auricular contractions is a prime factor in determining the rates of response. It has been determined experimentally, that if conduction is defective and the auricles are forced to beat more rapidly, the ventricular rate falls ; the ratio is increased not only because the bundle fails to transmit impulses at the faster rate, but because acceleration lowers the conduction power.

* First recorded in Fig. 79 of my " Lectures on the Heart " (*474*). White and Stevens and others have also recorded response to each auricular beat (*34, 676, 770*).

The degree of block is always patent in electrocardiographic records, unless the ratio is as 2 : 1 ; in this circumstance, alternate auricular complexes may be so fused with those of the ventricle that they are difficult to decipher (see *736* and redescription of same case in *457*) ; but, generally speaking, the continuous activity of the auricle, represented by a zig-zag or undulating line, can be clearly traced throughout the whole curve, though it is broken or deformed by ventricular deflections which are superimposed upon it.

When the ventricular responses are irregular, and this is frequently the case, the relative lengths of the ventricular cycles in flutter and in simple *A-V* block, are governed by the same law. A long pause permits recovery of conduction and at its termination the *As-Vs* interval is consequently short ; a short pause is succeeded by a response in which the *As-Vs* interval is widened. The arrangement of auricular and ventricular beats and their inter-relation is shown in Fig. 259 and 260. The first two responses in

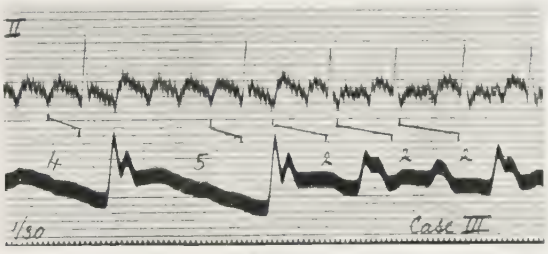


Fig. 259.

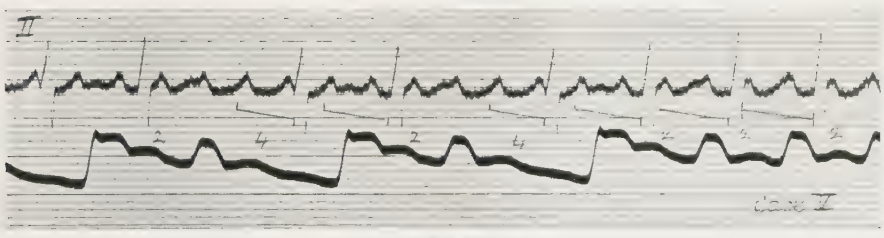


Fig. 260.

Fig. 259 and 260. (*Heart, 1912-13, IV, 171, Fig. 36 and 37.*) Each photograph shows an electrocardiogram and radial curve. Auricular flutter to which the ventricle is responding irregularly is present. The auricular systoles, responsible for ventricular contractions, are indicated diagrammatically. The numerals placed above the arterial beats affirm the number of auricular contractions to the corresponding ventricular cycles. Time in thirtieths of a second

Fig. 259 are from the auricular beats immediately preceding the ventricular contractions. Not so the third response ; the auricle completes a cycle and, while its impulse is on its way to the ventricle, begins and half accomplishes a second cycle. That two auricular impulses, one of which is to be effective while the other is to be ineffective, may be travelling towards the ventricle at the same moment, is witnessed to by this figure and with

equal distinctness by Fig. 260. When the auricular rate is extreme and 2 : 1 heart block prevails, the response of the ventricle is not to the auricular systole which comes immediately before it, but to that which falls with the preceding ventricular contraction (*457*).

Arterial curves.—The arterial curves of flutter are often distinctive of the condition when the responses of the ventricle are irregular (*457*). They illustrate in a very beautiful manner the principles of what is termed "spacing."

In flutter the heart is dominated by an auricular rhythm which is remarkable for its regularity. Whenever an arterial pulsation reaches the wrist, its position in time is controlled by a given auricular impulse standing in a perfectly regular series. The time interval between an auricular beat and the ventricular response (with its corresponding pulse) is strictly governed by the state of conduction at the instant and this in turn is governed by the period of preceding rest. *If two pulse beats are preceded by long pauses or by pauses of exactly equal lengths*, however close together or however far apart these beats may stand in a curve, the interval between them, and the interval separating the two auricular contractions responsible for them, are identical in length. And each interval of the kind represents the interval between *two* adjacent auricular contractions or a precise multiple of it. If the responses of the ventricle are at first to each second and later to each fourth auricular contraction, the later pulse rate is precisely half the original rate (Fig. 261*d*). Flutter curves may be subdivided into a number of lengths, subtending *chosen* pulse beats, all of which have simple arithmetic relations to each other. When a whole pulse curve may be sub-divided in this fashion, it is certain that the same dominant rhythm controls it throughout ; and in all such curves phases of irregularity are accurately repeated from time to time. It does not follow that individual pulse cycles will have a simple arithmetical relation to each other ; but those which are preceded and succeeded by equal conduction intervals, will bear this relation to each other and to stretches of the curve which begin and end in the same fashion. If a given pulse cycle is preceded by a shorter *As-Vs* interval and succeeded by a longer one, the cycle measures more than the corresponding number of auricular cycles ; if it is preceded by a longer and is succeeded by shorter *As-Vs* interval, the cycle measures less than the corresponding number of auricular cycles. The lengths of the conduction intervals vary inversely with the lengths of the preceding pauses. A long pulse cycle which is preceded by a short one consequently measures less than the corresponding number of auricular cycles ; conversely, a short cycle which is preceded by a long one measures more than the corresponding number of auricular cycles.

The analysis of the arterial curves is illustrated by the accompanying curves, all of which were taken from patients in whom the flutter had been determined electrocardiographically. In practice, the individual cycles in a given curve are compared by measurement, and above each is written the number of auricular cycles to which it is supposed to correspond. The

rules which govern this numbering are simple. Runs of short regular cycles are assumed to be at the same rate as the dominant rhythm, or at a simple multiple of it; they are numbered alike. The longer pauses of the curve are then compared with these, and they fall into two categories.

(*a*) Those which are of a length which is a precise multiple of the auricular cycles (calculated from the rapid beats). They are numbered correspondingly (see Fig. 261*d*).

(*b*) Those which have not these precise lengths. It may be that a cycle is longer than four calculated auricular cycles and shorter than five. If from a consideration of the pauses and the corresponding conduction intervals, shortening is to be anticipated, the higher number is adopted (Fig. 261*a*), if lengthening is expected, the lower number is used. Each cycle being thus numbered, the curve is subdivided into sections each containing a number of pulse cycles. In a case of flutter many such sections will be found of equal length; these will be bounded by beats which have equal pauses preceding them and will each contain the same calculated total of auricular cycles (Fig. 261*a-d*).

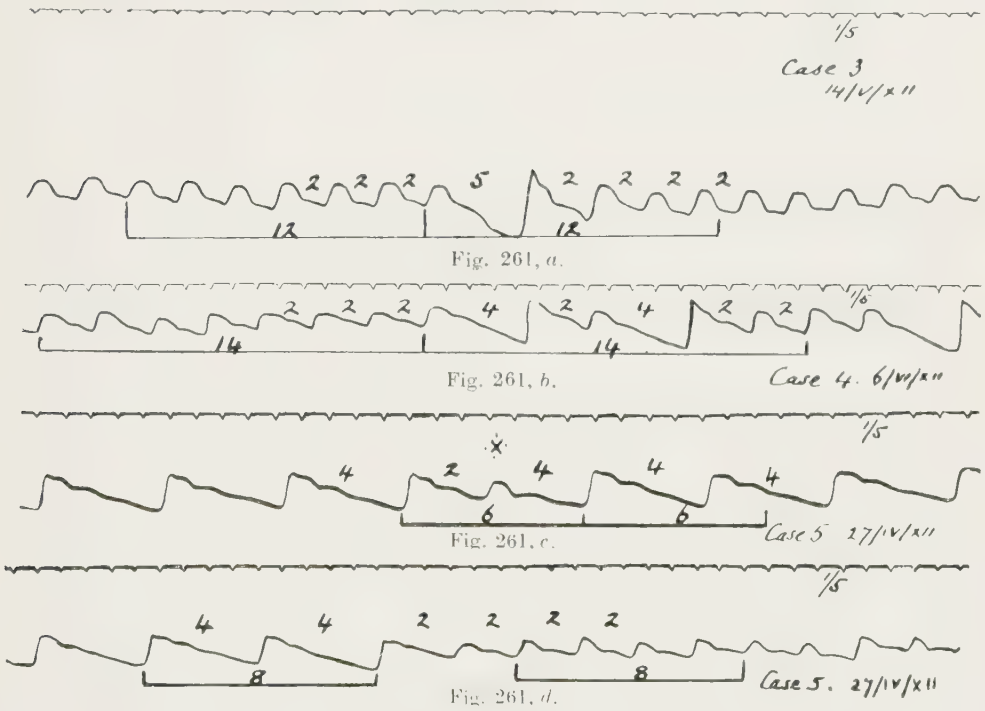


Fig. 261*a-d*. (*Heart, 1912-13, IV, 198, Fig. 1-4.*) Four arterial curves from cases of auricular flutter. They are published to show the methods adopted in analyses of these curves. The number of auricular cycles to the ventricle cycle is marked above the respective pulse beat. The bracketed portions of any single curve are of equal length; the number of auricular systoles corresponding to the beats included in a bracket is marked on the bracket. These analyses were checked electrocardiographically

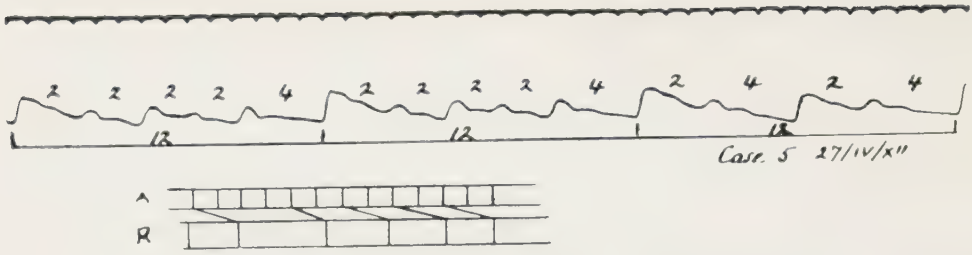


Fig. 262. (*Heart, 1912-13, IV, 199, Fig. 7.*) An arterial curve from a case of flutter. The pulse is very irregular and bears a superficial resemblance to that of auricular fibrillation. Note the presence of alternation. An explanatory diagram, in which the relations of auricular contractions and radial upstrokes are illustrated approximately, accompanies this figure. This analysis was checked electrocardiographically.

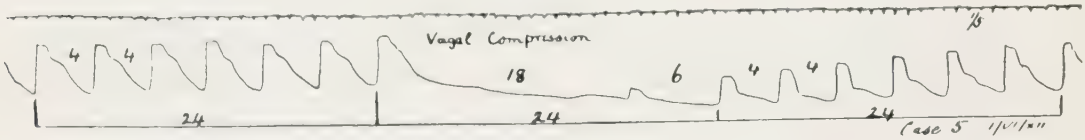


Fig. 263. (*Heart, 1912-13, IV, 201, Fig. 15.*) (× $\frac{2}{3}$.) An arterial curve showing the effect of right vagal compression during a period of 4 : 1 heart block. The auricular rate is known to have been unaffected because the ventricular responses, after the long pause, occur at anticipated points. From the same patient as Fig. 258.

The control of the irregularity by an unchanging dominant rhythm is well illustrated by vagal stimulation, an example of which is shown in Fig. 263. Upon compressing the vagus a regular 4 : 1 response is disturbed by two pauses, corresponding together to 24 auricular cycles. This correspondence is precise ; the ventricular beats, returning after stimulation fall at intervals which continue the original series.

The arterial curves in flutter are complicated by two circumstances. First, when the rate of ventricular response is high, alternation occurs (Fig. 262) and, secondly, responses to the auricle are so frequent that many of the pulse beats are weak and resemble extrasystoles. The strength of a pulse beat does not depend to any great extent upon the point of origin of the ventricular contraction which is responsible for it ; it is controlled mainly by the length of pause preceding it. A short pause is succeeded by a weak pulsation whether that pulsation is a response to the auricle, or is premature because it is extrasystolic ; the two types are indistinguishable in arterial curves ; the relation of the precise lengths of the cycles to one another alone enables their natures to be determined.

The relation between flutter and fibrillation, upon which the present therapy of flutter is based, is described and discussed in Chapter XXVII ; the nature of flutter is further discussed in Chapter XXVIII.

Chapter XXIII.

Auricular Fibrillation (The Clinical Condition).

In this and the succeeding chapter is an account of a specific clinical condition, which has come to be called "auricular fibrillation." In describing it I purpose to depart from the arrangement hitherto adopted, in which a description of experimental work precedes that of clinical observation. My chief reason for this course is that it permits me to present the subject, as it originally presented itself, namely, as a clinical problem, and to show how the solution gradually unfolded itself and how by persistent enquiry convincing evidences were at length obtained as to the true nature of this important disorder of the human heart.

As we now recognise it in man it is characterised by a single chief quality, namely, the absence of all signs of normal auricular contraction; further, it is responsible in the great majority of patients in whom it is found for a complete irregularity of the ventricular action and of the arterial pulse.

The irregularity, which is one of the chief features of the condition, is the commonest persistent irregularity exhibited by the human heart, constituting as it does approximately 50 per cent. of all such cases. It will be demonstrated that this disturbance of ventricular rhythm is to be sought in the auricle and attributed to temporary or permanent fibrillation of this chamber. This conclusion and our detailed knowledge of the condition is due to the work of a very large body of men. Fully possessed of the facts, we may now trace the earlier work along two independent paths. Observations were undertaken upon the arterial pulse; others were carried out upon the venous system; each series being distinct and for very many years unassociated with the other. The two paths of investigation converged and finally met in modern times.

On the one hand, a conspicuously irregular arterial pulse, especially associated with mitral disease in its later stages, was the subject of study by mechanical means from the time of the introduction of the sphygmograph. It is portrayed by Marey (*536*), Riegel (*621*, *622*), Sommerbrodt (*702*), and many other writers. It has been termed the "mitral pulse," and has been attributed to "delirium of the heart," amongst other causes. It has passed by the names *pulsus arrhythmicus* and *pulsus irregularis* (*622*), and has been identified, in a classic but obsolete nomenclature, with the adjectives *irregularis*, *inæqualis*, *deficiens* and *intermittens*.

On the other hand, a prominent *systolic* pulsation in the veins of the neck was described (*16*, *701*), and was attributed to tricuspid incompetence. This timing of the venous pulsation was endorsed by Riegel, who obtained the first graphic records of the movement; but the class of cases in which

such pulsation is essentially found was not isolated, neither was its full significance grasped, until the more exact and more applicable technique of Mackenzie was introduced.

It is since the year 1902, when "*The Study of the Pulse*" was published (*501*), that chief progress has been made. To Mackenzie we owe the correlation of two phenomena, gross irregularity of the heart and the systolic venous movement, which he has termed the "ventricular form of venous pulse." In the work referred to, this writer first demonstrated their frequent association and ascribed them both to a single underlying condition, namely paralysis of the auricle. A year later, Hering (*257*, *274*), describing the arterial pulse alone, laid more stress upon its characteristics and spoke of it under the title *pulsus irregularis perpetuus*. The conclusion that it is a condition *sui generis* came slowly and mainly through the efforts of Mackenzie; it was a conclusion of prime importance, being the first chief step which led to the discovery of the underlying mechanism. Mackenzie laid stress upon the frequent association between irregularity and the ventricular form of venous pulse in 1904 (*503*, see also *272*), and the prominence given to this association in a later paper, based upon 500 patients (*510*), is to be emphasized. The final demonstration that gross irregularity of the ventricular action is of a specific nature was obtained electrocardiographically (*434*).

In his papers of 1904-5, Mackenzie brought forward several new and important facts and most striking amongst them, in the light of our present knowledge, was evidence that the auricle is active. Formerly regarding the auricle as paralysed, because no sign of its activity could be found, and considering the rhythm to originate in the ventricle (*508*), he attempted to separate a special group of cases in which auricular activity might be considered probable. Auricular activity was assumed, because the auricle was found hypertrophied at autopsy (*507*); and because cases were observed in which the normal rhythm reasserted itself (*503*). It is to these papers that we are more especially indebted for the observation that in no case of complete irregularity of the heart are there signs of the *normal* auricular contraction during diastole and, further, for the first record of cases of this nature, in which it is probable that little dilatation of the right heart and little tricuspid regurgitation is present (see also *725*). His earlier view that the condition resulted from auricular distension as a consequence of valve incompetence was at least partially abandoned, and the rhythm was ascribed as the cause rather than as the result of cardiac dilatation. In 1904, Mackenzie postulated the view that in some cases the seat of the rhythm may lie in the junctional fibres lying between auricle and ventricle, and, by conceiving the simultaneous contraction of auricle and ventricle in response to impulses from this single source, attempted to explain the absence of all sign of normal auricular contraction which he had demonstrated to be one of the chief features of such cases.

In 1907-8, Mackenzie (*510*, *511*, *515*) adopted the provisional hypothesis of the nodal origin of the rhythm more generally, holding the auriculo-

ventricular node to be the seat of disturbance in all cases of irregularity found in combination with the systolic form of venous pulse. He therefore included all such cases under the term "nodal rhythm." This conclusion was abandoned, when undoubted cases of "nodal rhythm" (see Chapter XX) were described, and when other evidences of clinical fibrillation of the auricle were placed on record.*

The clinical condition.

The radial pulse curves.

The character of the radial pulse curves in complete irregularity of the heart is so striking that it could not, and as we have seen did not, escape early attention.

The irregularity is of the most varied description. The pulse may be slow or fast, and the variation in rate is great (30 to 200 per minute). All the beats may be of small excursion; more commonly there is a haphazard intermingling of forcible and weak contractions, and the latter are often conspicuously dicrotic. The radial pulse is but an indifferent index of the rate of the ventricle; many beats are not transmitted. The pulse rate may be considerably reduced, either as a result of these abortive contractions or as a consequence of the actual slow speed of the ventricle. The beats may show coupling over short or long stretches of curve. The fast types are the commonest, and in these the usual rate of the ventricle is approximately double the normal rate (110 to 150). It is usually at these fast rates that the disorderly character of the pulsation is prominent. With the slower rates the irregularity is less evident. In arterial curves the disorder may be recognised by two criteria. First and most important is the absolute character of the arrhythmia. The heart action is never regular, and seldom do two beats of the same character or length occur in succession. In a long curve, it is rare to find any two short sections of tracing which possess even a superficial resemblance to each other. The pauses between the beats bear no relation to one another, and in this feature the irregularity stands in contrast with all other varieties. The second criterion consists in the lack of continued relation between the strength of a beat and the length of the cycle which precedes it. A strong beat may follow a short pause, and a weak beat may succeed a long pause. A few examples of the pictures presented by Dudgeon tracings are given in Fig. 264. They may serve, with the brief notes attached to them, as a guide in recognising the type of case with which we are dealing. They illustrate the main points referred to in the text, but the variety shown is so great that they can scarcely be held even as representative of the irregularities which may occur. Numerous and additional examples are scattered throughout the simultaneous tracings which illustrate this and the succeeding chapter.

* An historical account of the discovery of auricular fibrillation is to be found in the British Medical Journal (*452*).

The venous pulse curves.

"The ventricular form of venous pulse" is a term which expresses the only fixed quality manifested by graphic records taken from the jugular veins in these cases. It implies that all prominent and rapid changes of

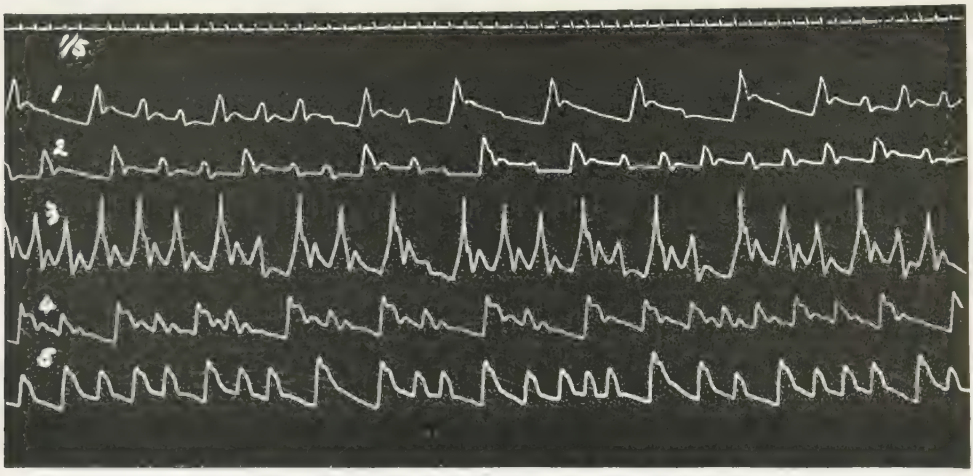


Fig. 264. ( ) (*Heart, 1909-10, I, 315. Fig. 1.*) Radial pulse curves taken with a Dudgeon sphygmograph. The time tracing, which applies to all curves beneath it, is in fifths of a second. The figure illustrates the chief features of complete irregularity of the pulse.

1 and 2. From a man aged 48, admitted to hospital suffering from mitral stenosis of rheumatic origin and general cardiac dilatation. Enlargement of liver, distended veins and dropsy were present. Irregularity complete and persistent; apical murmurs early and mid-diastolic; venous curves of ventricular form; electrocardiographic curves of usual type.

3. From a man aged 64, the subject of bronchitis, emphysema and arteriosclerosis. No history of rheumatism. Heart somewhat enlarged to right and left. Heart sounds normal; S. B. P., 150 mm. Hg..* With the exception of shortness of breath on exertion no signs of heart failure were present. The irregularity disappeared on one occasion for a few days and the pulse regularity was then interrupted by auricular extrasystoles. The *a-c* interval was normal. With the complete irregularity the venous pulse was ventricular in outline, the electrocardiogram was typical.

4. From a man aged 37, suffering from mitral stenosis of rheumatic origin. Heart enlarged to right and left, dyspnœa and slight liver enlargement, no dropsy. Pulse persistently irregular; ventricular form of venous pulse; electrocardiograms typical.

5. From a man aged 65, suffering from aneurismal dilatation of the whole thoracic aorta, pulmonary œdema, associated with arterial sclerosis, emphysema and signs of sclerotic kidney. Dropsy and liver enlargement present. Pulse persistently irregular; ventricular form of venous pulse. Died unexpectedly.

* This blood-pressure reading is a measure of the obliteration pressure of the most forcible beats. Blood-pressure estimations in cases of complete irregularity are extremely unsatisfactory; the beats force their way through the armlet at widely varying pressures.

volume in the venous cistern fall within the limits of ventricular systole. The curves corresponding to the individual heart beats vary in their positions relatively to each other just as do the radial beats. There may be considerable variation in the amplitude of the separate curves in a given case. This variation is certainly not instrumental in origin, for close examination reveals the recurrence of a particular type of curve after a given interval of rest, or during a given phase of respiration. As a general rule, and in a single case, a large venous beat accompanies a large radial beat, but the difference in size from one beat to the next is less in the former than in the latter. A family resemblance between the separate venous beats of a single curve is generally if not always present.

The venous curve corresponding to a single heart cycle is generally composed of two or three peaks, and a similar number of dips. The upstroke of the first peak is synchronous with the beginning of the carotid pulsation at the same level of the neck (though it may precede or succeed it slightly). The downstroke of the last peak starts at a point corresponding in the neck to the opening of the tricuspid valves. It is synchronous with the bottom of the downstroke of the cardiogram, or with a point a little later than the bottom of the dicrotic notch on the carotid tracing. The chief depressions follow the first and last peaks and are very variable in degree from case to case. As a general rule it may be said that the shorter the duration of the abnormal mechanism the deeper is the first as compared with the second depression, and that in old-standing cases the dip in the centre of systole is replaced by a larger and fuller complex of systolic peaks. There is a relation between the mean distension of the veins and the swelling of those veins in systole. Thus, in cases of long duration, in which the veins are much dilated, the venous curve is in the form of a prominent systolic plateau. The older conception, that the prominence of the venous pulsation is an index of the degree of tricuspid reflux, is probably not without some foundation. The curves obtained from patients soon after the onset of the disorder and the curves in cases in which the heart's engorgement is but slight are similar, in their systolic phases, to the curves from normal subjects (Fig. 265*b*). In cases where the heart is engorged and the veins overfilled, the first depression (corresponding to x in normal curves) becomes more or less completely filled (Fig. 265*a*) until in advanced conditions of venous stasis the curve assumes the plateau form (Fig. 267*a* and 269); in these circumstances it has a flat top and resembles the curve of intraventricular pressure. The transition from one type to the other may be followed from case to case, or in a single case. The flat topped curve often accompanies rapid ventricular action for then overfilling of the veins is the rule.*

* The plateau form of venous pulse found in many cases of fibrillation is attributed by Niles and Wiggers (*575*) to defects in the method of recording and to impact waves from the carotid. To this opinion I cannot consent, although I fully realise the limitations of the method adopted. The plateau form can be recognised clinically by watching the sustained swellings of the vein in systole or by projecting the shadow of the vein directly on to a recording surface. This form is not at all uncommon.

Fig. 265. Polygraphic curves from cases of clinical fibrillation of the auricles. The arterial curve is in each case grossly irregular: the venous curves are of the ventricular form.

The relation of the normal type of curve to the various forms of ventricular venous pulse curve is diagrammatised in Fig. 266. The outlines have been drawn from actual curves. The dotted outline is that of the auricular form of venous pulse. The diagram displays transitions between various types of the ventricular form of venous pulse. But the distension of the auricle and the deformation of the venous curve may take place, while, during the transition, the presystolic auricular contraction is present, or while, during the transition, the co-ordinate systoles of the auricle are suspended. The ventricular form of venous pulse may be conspicuous even in its plateau form, while the normal heart sequence is maintained (see Fig. 100, page 151), and, as we have already seen, the usual or normal systolic portion of the venous pulse may be found (the type with the deep x' depression) and yet the signs of co-ordinate and presystolic auricular contraction may be entirely in abeyance.

The two phenomena, the sudden disappearance of all signs of normal auricular activity on the one hand, and venous engorgement on the other, are not necessarily associated; either may appear without the other. The virtual palsy of the auricle is not responsible for material venous stasis (*456*),

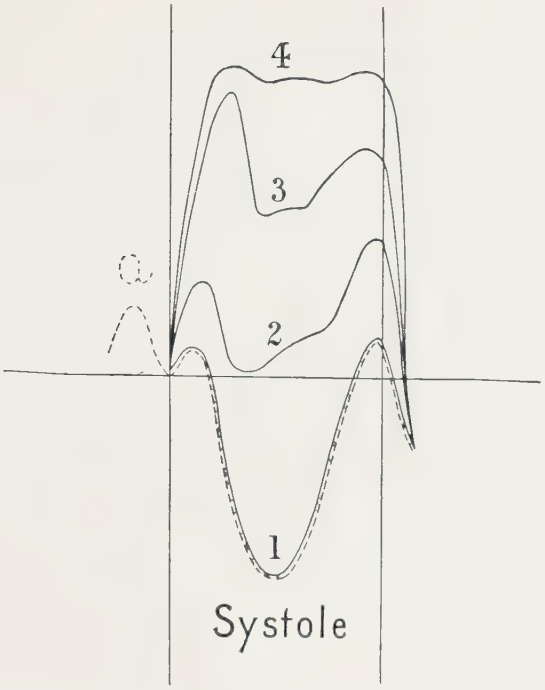


Fig. 266. (*Heart, 1909-10, I, 320, Fig. 3.*) A diagram illustrating the relation of the auricular and ventricular forms of venous pulse, and the commoner variations in shape to which they are liable. The dotted line represents the physiological type, the auricular form of venous pulse. The continuous lines represent the ventricular portions of types of curve met with when either the auricular or ventricular forms of venous pulse are present in patients.

though it may presumably aggravate it in a small measure*; conversely, venous stasis is not the cause of the auricular paralysis in these cases. The diagram illustrates the transition of the systolic portion of either auricular or ventricular form of venous pulse curve from the type with perfect to the type with imperfect venous flow. In brief, it may be said that there is but one constant distinguishing mark between auricular and ventricular forms of venous pulse, and it consists of the auriculo-systolic or *a* wave in the former. Individual cycles of a curve, which is an example of the ventricular form of venous pulse, can be identified as such only by noting the absence of this wave.

Certain waves which occur in diastole.

It has been said that the ventricular form of venous pulse is composed of waves of which the most prominent and abrupt occur in systole. Diastolic waves are also seen. The auriculo-systolic wave does not occur, but any of the remaining waves seen in the diastoles of normal heart cycles may appear. Thus, it is common to notice a gradual or even abrupt rise of the line as

* Gesell (*224*) believes that the auricles play a greater part (by filling the ventricles) in maintaining arterial pressure than I feel prepared to allow.

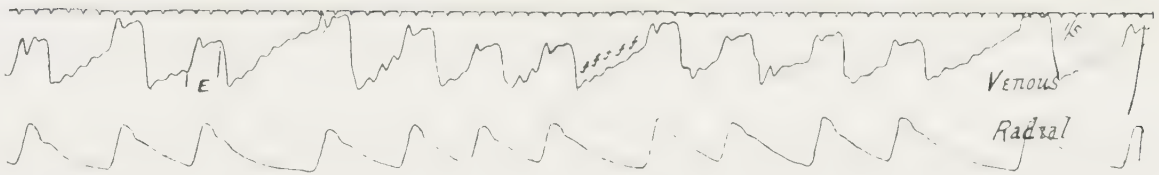


Fig. 267*a*.

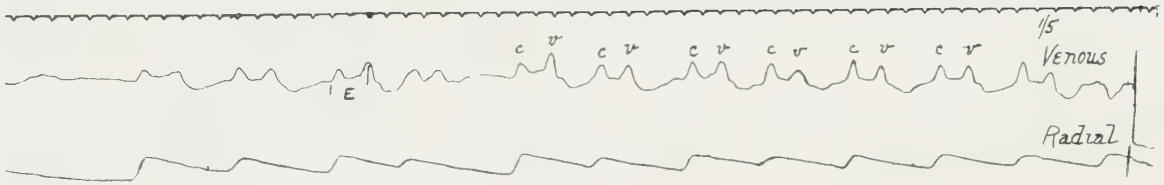


Fig. 267*b*.

Fig. 267*a* and *b*. ($\times \frac{2}{3}$.) Two polygraphic curves from cases of auricular fibrillation, displaying complete irregularity of the ventricle and the ventricular form of venous pulse. In the top curve the systolic elements are of the plateau form, while in diastole the stasis rise of the curve is complicated by an almost regular series of small undulations *f*, *f*. *f*.

diastole proceeds, and this is due to filling of the veins when the ventricle has admitted all, or the greater part, of its content of blood ; this "second onflow wave," as Morrow (*565*) calls it, is most evident in long diastoles and when average venous pressure is high. It is seen to advantage in Fig. 267*a* ; the curve rises as the veins swell in the long diastoles.

But the most important waves of the diastole are undulations, the cause of which will be discussed in the next chapter. These are rapid and, as a rule, small oscillations, often remarkably regular in amplitude and sequence and often filling the whole stretch of diastole. By no means always present in clinical records, they are of sufficient frequence to be of practical value, for they are found in no other condition. They are well displayed in Fig. 267*a* (*f*, *f*, *f*). It is in cases when the ventricular action is slow that they are most prominent ; from 350-500 per minute is the measure of their frequence. First described by Wenckebach (*761*), and later by Mackenzie (*510*, *511*),* they have since been recorded by many workers. In some patients, and especially in those in whom the ventricular rate is relatively rapid in the absence of much venous engorgement, these waves are often coarser and, occurring throughout systole as well as diastole, convert the venous curve into a series of rapid and irregular oscillations which preclude any detailed analysis of the venous curve as a whole ; an example of such a curve is given in Fig. 265*d*.

The electrocardiographic curves in limb leads.

The first electrocardiogram which can be identified as one of clinical fibrillation of the auricles was published by Einthoven (*111*) ; but it was not until I published a series of these remarkable records, simultaneously with those of Viennese workers (*430*, *434*, *437*, *442*, *660-662*), that the significance or clinical associations were appreciated. We are now possessed of innumerable published examples (*275*, *280*, *345*, *355*, *390*).

The curves consist of ventricular complexes arranged in an irregular sequence corresponding to that of the ventricular beats. The ventricular complexes consist of initial deflections *Q*, *R*, *S* and a final deflection, *T*. The individual ventricular complexes do not differ from those associated with normal beating of the chambers, they show the same variations as do the latter. Thus those changes which are associated with preponderating hypertrophy of one or other ventricle, obscurity of *T* or its inversion, and more rarely aberrant types of ventricular complex, are also seen from case to case ; but the predominant type is that which displays ventricular complexes of normal outline ; the curves are those of supraventricular type.

A comparison of the height of the summits *R* and the strength of the corresponding pulse beats, shows that there is no fixed relation between

* In these two papers Mackenzie actually ascribes the waves to auricular fibrillation, though it is not clear how he conceived this disturbance to be simultaneous with "nodal rhythm." But in subsequently describing the same curves in his book (*515*, *1st edition*, *page 299*), he states his belief that the waves were produced artificially and were not due to fibrillation of the auricles.

them : neither is there a fixed relation between the amplitude of R and the diastolic pause which precedes it.

The most significant features of the electric curves are the absence of the presystolic deflection P (*275*), which accompanies all normal heart beats, and the replacement of these summits by a series of oscillations of

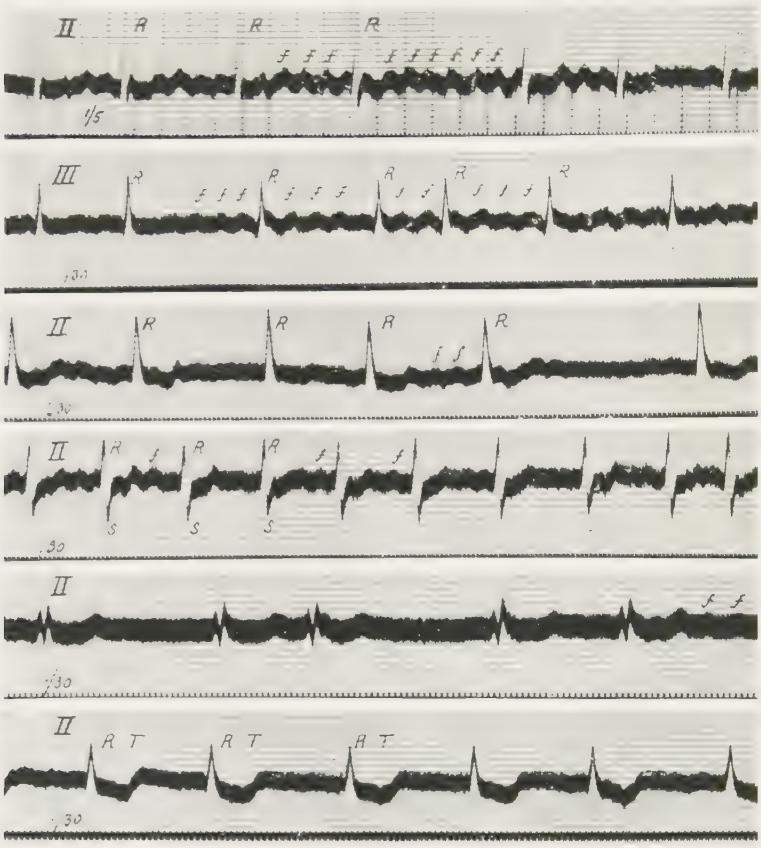


Fig. 268. ($\times \frac{4}{5}$.) Six electrocardiograms from separate patients, illustrating some of the chief forms of curve obtained in cases of clinical fibrillation of the auricles. These curves present certain constant features. In the first place, the ventricular complexes are of constant form in any one of these curves. In the second place, they lie at irregular intervals. In the third place the initial phases are of short duration and have the general characters which beats of supraventricular origin display. In the fourth place, in none of the curves do P summits occur, though here and there these may be simulated by a single oscillation. In the fifth place oscillations are present. These oscillations are conspicuous in the first and second curves and are of characteristic form and frequence, being of irregular outline and amplitude and fading away from time to time. In the third and fourth curves they are less conspicuous though easy to identify. In the fifth curve they appear only in the last diastole; in the last curve, selected to illustrate this point, they are so inconspicuous as almost to escape notice.

In the first curve the time is marked in fifths and thirtieths and in the remaining curves in thirtieths of a second.

greater or lesser amplitude (*430*, *660*). These oscillations are variable both in prominence, shape and frequence from case to case and in one and the same case. In some curves, and especially in cases of mitral stenosis, they are so large that each has an amplitude as great or even greater than that of a natural *P*. However large they are (and amplitude of the oscillations is in general related inversely to their frequence) *they are never quite regular in form, amplitude or spacing* in the condition I am describing, namely, cases in which there is complete irregularity of the ventricles. In other curves they may be far less conspicuous and may be distinguished only from point to point.* For considerable stretches they may be absent; nevertheless they are constantly present in cases of complete irregularity of the heart; if undiscovered in one lead they are apparent in another. As a rule leads *II* and *III* display them best. They occur also during the period of systole, and falling with and being superimposed upon *T* frequently distort it, sometimes past recognition. The clean cut character of *R* is not affected owing to the rapidity of its movement. Most conspicuous in long diastoles,

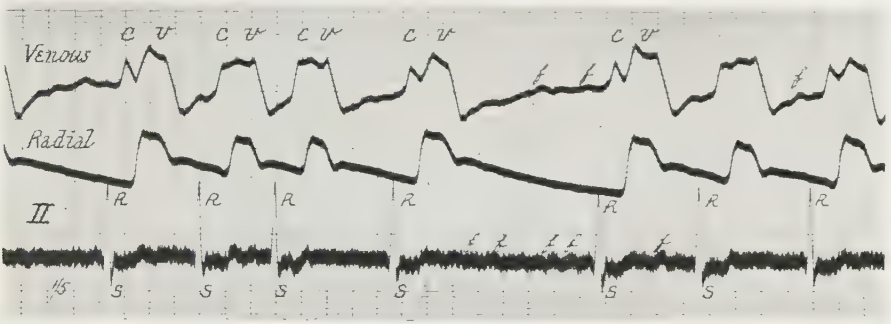


Fig. 269. ($\times \frac{3}{4}$.) Simultaneous venous, radial and electrocardiographic curves form a clinical instance of fibrillation of the auricles. The venous curve is of the plateau form and shows the undulations *f*, *f*, in the longest diastoles. The radial curve displays the gross pulse irregularity. The electrocardiogram is characteristic; ventricular complexes of supra-ventricular type are scattered irregularly throughout the curve; in the diastoles oscillations replace the customary *P* summits. Time in fifths of a second.

they are seen to best advantage when the ventricle beats slowly; when the ventricle beats rapidly, the ventricular complexes are close set and the oscillations fall chiefly within the confines of systole; in these circumstances their presence is known because they distort *T*. Those sections of the curve which lie between adjacent *R* summits consequently vary in form, and give to the curve as a whole a peculiarity at once characteristic and unmistakable.

* There has been a recent tendency (*167*) to speak of the coarser oscillations as portraying flutter and not fibrillation. It may be that in certain of the coarse waved curves the condition present in the auricle is a less profound disturbance and more closely allied to flutter than is the fine waved variety, but it is not flutter, properly so-called, and the application of the term flutter to such clinical cases is only confusing (see remarks on page 328.)

Complete irregularity of the heart is the result of auricular fibrillation.

The irregular oscillations seen upon galvanometric curves are due to an incoordinate contraction of some portion of the heart; they are not a direct result of structural change in the heart, and are independent of movements of the somatic musculature.

The oscillations are independent of structural change in the heart;* they have no relation either in their amplitude or frequence to the extent of the muscle changes. They are absent in cases of gross myocardial disease when the normal sequence of chamber contraction is maintained. On the other hand, a patient who exhibits no sign of gross myocardial disease, in whom the heart is not dilated and in whom there are few symptoms of defective circulation even upon exercise, may show these oscillations to perfection. The oscillations appear when the ventricular action becomes irregular (*434*); they come when the normal summits *P* disappear from the curve. Their presence is clearly associated with these two events.

The curves of Fig. 270 and Fig. 271 were secured within a few days from one and the same case. The curves of Fig. 270 are from the three leads of the patient during a period when the heart's action was perfectly regular. A day or so later the patient returned in an attack in which the heart's action was grossly and continuously irregular. The curves from the three leads are shown in Fig. 271. Comparing the two series we may note the similarities and the differences. In corresponding leads of the two series the characters of the ventricular deflections are identical. In the one series the heart's action is regular, in the other irregular. In the regular series *P* is present and normal in form; in the irregular series it is not seen. In the regular series there are no oscillations, in the irregular series they are conspicuous. I cannot over-emphasise the importance of comparing series of curves from one and the same case at different periods, if the heart's mechanism is variable. Such collections of curves are invaluable. As an illustration of this fact the proof which the present figures afford that the ventricular complexes are of normal type during the period of disturbance may be cited, for they are identical with those obtained while this same heart was acting normally. Again, any question of the responsibility of myocardial change in producing the oscillations of the disturbed period, is at once excluded; for in this patient many such series were taken from regular and irregular periods which alternated, yet the curves were always of the forms shown. These,

* Though this may modify them, witness the larger oscillations in cases of mitral stenosis.

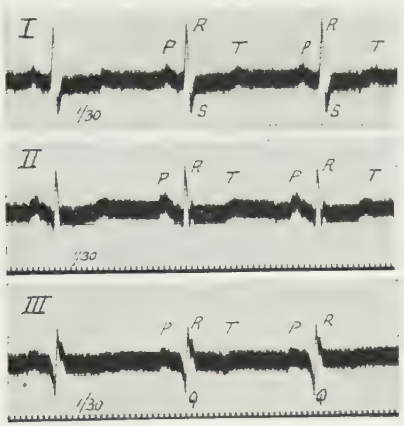


Fig. 270.

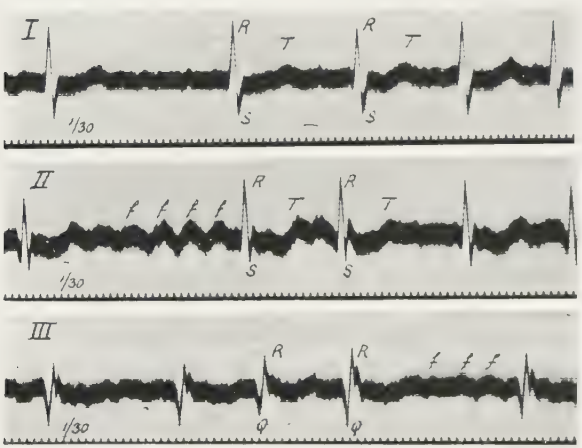


Fig. 271.

Fig. 270 and 271. ($\times \frac{4}{5}$.) Electrocardiograms from the three leads in a case of paroxysmal tachycardia due to fibrillation of the auricles. Fig. 270 was taken during a period of quiescence and the curves are in every respect normal. Fig. 271 was taken during an attack. The ventricular complexes are unchanged in type, but are placed irregularly. *P* does not appear, it is replaced by irregular oscillations *f*, *f*. Time in thirtieths of a second.

and similar series of curves, may also be used as evidence that the oscillations do not originate in the somatic musculature, but in the heart itself (*434*). It is true that electrocardiograms may now and again show oscillations of this form which may be ascribed to a movement of a muscle in arm or leg. But the oscillations which we are now considering bear no relation to limb movements such as are to be observed. In patients who display them from time to time, unusual muscular movements are not witnessed, the conditions in the somatic musculature are the same whether the oscillations are present or absent.

Fig. 272*a*

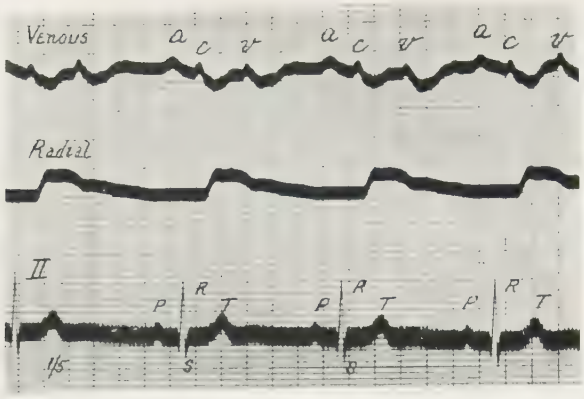


Fig. 272*b*.

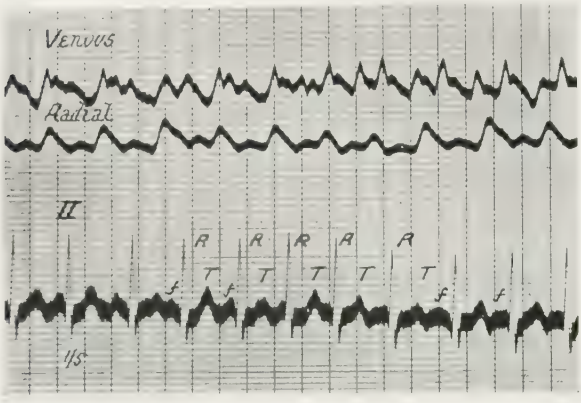


Fig. 272*a* and *b*. ($\times \frac{2}{3}$.) Simultaneous venous, radial and electrocardiographic curves taken from a patient who suffered from paroxysms of tachycardia, due to fibrillation of the auricles. In the periods of quiescence (Fig. 272*a*) the heart's action was regular and slow, the venous and the electrocardiographic curves demonstrated a perfectly normal heart mechanism. During the attacks (Fig. 273*b*) the ventricle beat rapidly and irregularly. The venous curve is so distorted by fine and coarse undulations that its analysis is scarcely possible (compare Fig. 265*d*). In the electrocardiogram the appearances are characteristic in that there are no *P* summits, but oscillations (*f*, *f*) spring up in the short diastoles and also fall with the *T*'s and distort them. Time in fifths of a second.

Muscular movements give rise to irregularities in the curves when a patient trembles or fidgets. In the great majority of such cases these extraneous vibrations can be identified at once by their general appearance and rate. If sufficient precautions are taken no such irregularities appear in subjects in whom the heart sequence is normal. Oscillations are invariably present in the class of patient considered, whatever the precautions adopted. They are of much the same degree from day to day and from hour to hour in the same subject. They are prominent when arm-leg leads are adopted,

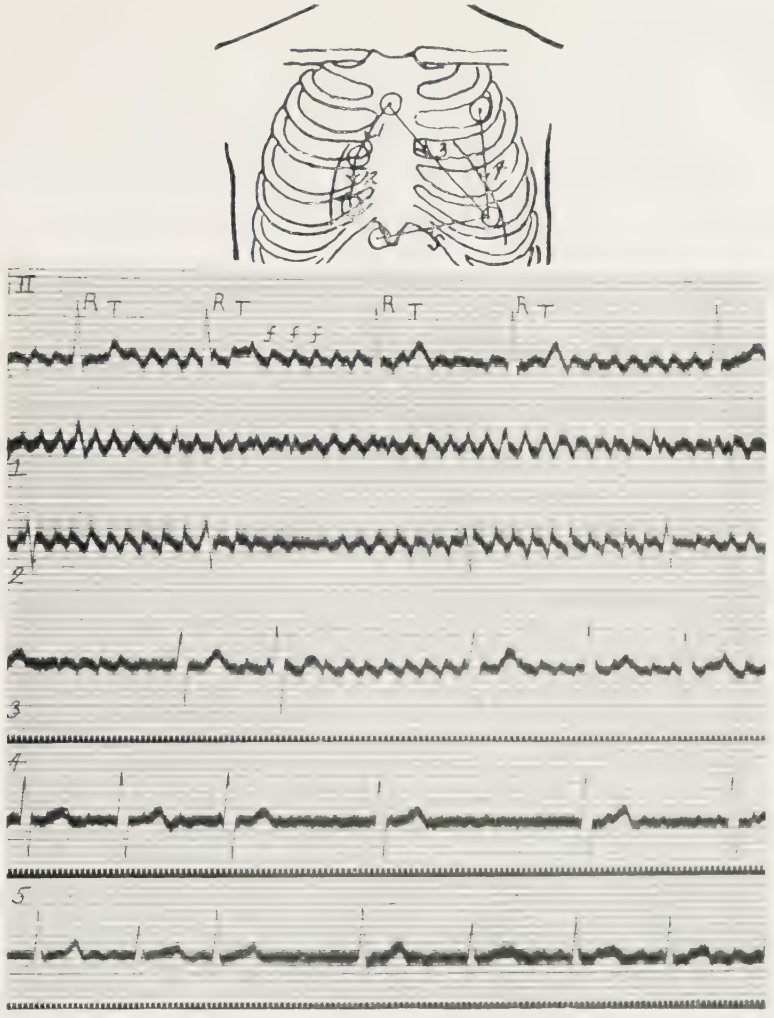


Fig. 273. A diagram of the chest wall showing the special leads (*1* to *5*) used in analysing the curves of auricular fibrillation; also six electrocardiograms. The first electrocardiogram is from lead *II*; it consists of irregularly placed ventricular complexes (*R*, *T*) and of large and continuous oscillations (*f*, *f*). The remaining five curves are from the chest wall. *1* and *2* were taken from the area overlying the right auricle; in these leads the oscillations are maximal and the ventricular complexes are minimal. *3* was taken from an oblique lead covering the whole heart, and it shows both oscillations and ventricular complexes well. *4* and *5* were taken from leads along the margins of the ventricles; they show but little sign of the oscillations. From a case of mitral stenosis. Time in thirtieths of a second.

but vanish almost completely if the electrodes are attached to the two inferior extremities. They are almost always inconspicuous when the arms are paired to form a lead.

Numerous and special leads have been devised and have been employed to exclude their origin from abdomen, limbs, head and neck. It is a matter

of indifference, so far as the amplitude of the oscillations is concerned, as to how much somatic muscle is between the contacts. The amplitude is controlled by the proximity of the lead to the heart. They emanate from the heart, and, as we shall see, from a particular part of this organ; they appear simultaneously with other phenomena, signs of virtual paralysis of the auricle and gross irregularity of the ventricle. The electrical disturbance so portrayed signals an event which is an essential part of the changed heart mechanism.

The oscillations arise in the vicinity of the auricle. The excitation wave travels to the ventricle along the normal paths of conduction.

The origin of the oscillations in electrical curves may be shown by using special leads from the chest wall (*434*). This demonstration is most striking in cases of complete heart irregularity in which the right auricle is distended, for in these patients a large surface of this chamber is in contact with the chest wall. I use discs of copper as contacts and fasten them to the chest by means of a stiff paste of flour, salt and water. As an illustration Fig. 273 is used. The top curve of this figure is a standardised electrocardiogram from lead *II* (right arm and left leg); it shows the prominent oscillations. The remaining curves are standardised curves from the chest wall, the actual positions of the contacts in five separate leads being shown in the diagram. The curve of lead *3*, from base to apex of the heart, shows the ventricular beats, and the oscillations are as prominent as in the limb lead. In lead *1* and lead *2* the contacts were placed in the vicinity of the right auricle; these curves consist almost exclusively of oscillations and these occur continuously. In leads *4* and *5* on the other hand, the contacts were placed along the borders of the ventricles and, whereas these leads show prominent ventricular deflections, the oscillations are absent or inconspicuous.

This example is but one of very many other patients similarly examined and yielding similar results. Such leads and those taken from other regions of the chest wall show that the oscillations are conspicuous or the reverse, according as the contacts are close to or distant from the auricle. In a patient who suffered from paroxysms of auricular fibrillation I have been able to show that the prominence of the oscillations in any given chest lead during the stage of the paroxysm goes hand in hand with the prominence of the auricular deflection in the same lead while the heart is beating normally (*449*). The source of the oscillations is thus traced to the auricular portion of the heart.

The method of leading I have described analyses axial curves into their auricular and ventricular elements and shows that in limb leads the two series of electrical changes, on the one hand a continuous series of oscillations generated in the auricle, and on the other the separate ventricular complexes, are superimposed and combined in the one curve. Those curves which are obtained from direct leads over the auricle, give the purest pictures of the oscillations,

which then show frequencies varying between 400 and 600 per minute. At all events this is the number of the chief oscillations to the minute : other finer and more rapid oscillations are sometimes present, whose frequence may be as high or higher than 2,000 per minute.

The direct leads may be used to obtain further evidence, if such is required, that the excitation wave spreads in the ventricle in a normal fashion during the heart's complete irregularity. We have already noted the resemblance between the ventricular elements of the curves in the three limb leads during the quiescent and paroxysmal period in suitable cases. The resemblance is indeed remarkable, and is extended even to the minute detail of the curves. A precisely similar statement applies to the ventricular complex taken from chest leads in and out of the attack (*449*). In any of these leads, nay, in a given lead from any part of the body, the shape of the ventricular complex is the same whether the heart beats normally or irregularly.

From these observations we may positively conclude that the spread of the excitation wave in the ventricle is along the normal paths of conduction when the heart is beating irregularly.

Conclusions from the clinical findings.

It may be well to summarise at this stage our knowledge of this irregularity of the human heart so far as it has been considered and to show the direction in which such knowledge leads us.

The irregularity is a complete irregularity, it is not repeated in phases, as is the case with all other forms of cardiac irregularity. This feature is in itself distinctive, in itself requires explanation. But although the irregularity is so complete, the beats of the ventricle, in uncomplicated cases, are all of supraventricular type ; each beat is propagated from an impulse which is derived from a supraventricular source, a source which lies above the level at which the bundle bifurcates. This is the first guide to the seat of disturbance, the ventricle is exonerated.

The second and constant characteristic of complete irregularity is that, whereas in normal subjects and in patients suffering from all other forms of heart irregularity or cardiac disability the auricular systole leaves a definite impress either upon the cardiogram, upon the venous volume curve, upon the œsophageal curves (*349*, *617*), or upon the electrocardiographic curve, such evidences of its normal activity are consistently wanting, whichever method of exploration is employed, in the group of cases which is now engaging our attention.

The conclusion, which it is impossible to avoid, is that the *normal presystolic* auricular contraction is in abeyance, temporarily or permanently.

Actual paralysis of the auricle was suggested, but little support is now found for this hypothesis. The conditions of the circulation are often such that it is impossible to suppose that the pressure in the auricles is increased.

The observation of hypertrophy in the auricle at autopsy led Mackenzie to abandon his earlier view of paralysis and converted him to the belief that the auricle is active. This belief was supported by the reappearance of signs of auricular contraction in paroxysmal cases. We are led to a precisely similar conclusion by the evidence yielded by the electrocardiographic curves. The auricle is the seat of an electric disturbance of a peculiar yet distinctive nature. The constancy of the oscillations, their unique appearance and *their presence throughout the whole of the cardiac cycle*, convinces us that they are an essential feature of complete irregularity and that *the activity of the auricle is continual.*

Co-ordinate contraction of the auricle at any period of the cardiac cycle other than that of ventricular systole can be readily excluded. Co-ordinate and simultaneous contraction of auricle and ventricle can also be set aside, for, as we have seen, it gives rise to totally different pictures.

In experimental work we encounter but one variety of auricular activity in which inco-ordination is present, and this mechanism is one in which the auricle is in unceasing movement. It is the state known as fibrillation.

The final proof that complete irregularity of the heart in man is the result of fibrillation of the auricle, depends upon a close comparison of observations in man and animals. This comparison is given in the succeeding chapter.

Chapter XXIV.

AURICULAR FIBRILLATION (*Continued*).

Auricular fibrillation as it is seen in experiment.

The auricles are easily made to fibrillate in the dog by submitting them to a faradic current. The effect of so stimulating the auricle was briefly described by McWilliam (*522*) in 1887; many years later it became a matter of more detailed study by Phillips (*596*), Fredericq (*187*), and others (*397*). When the auricle is so treated it passes into a condition of fibrillation. In this state the walls of the chamber stand in diastole (Fig. 274); systole is not accomplished; the wall as a whole remains stationary but for the movement conveyed to it when the ventricle contracts. Yet close attention to the muscle surface at once reveals feverish activity; the whole surface is alive with twitchings, or coarser undulatory movements, which in small adjacent areas appear to be independent of each other; the little movement, that is to say the local twitch or local undulation, seems not to be propagated to any considerable distance but to come into conflict with similar muscular movements in its neighbourhood.* Attempts to record the general movement mechanically are unsuccessful; at the most a tremulous line is written. This is the full condition which is properly termed *fibrillation*. It is explained by supposing that the auricular tissue is broken up into a number of areas, each of which is contracting independently of surrounding areas; but that is still hypothesis. At times the movement is coarser and then mechanical records may be obtained and show some tendency to regularity of the undulation; but when the movement is of this variety the condition is generally speaking unstable and at any moment the normal rhythm may be resumed. For the moment we may fix attention upon salient features. The disturbance is not confined to the auricle (*86*, *596*), it disturbs the harmonious beating of the ventricle; fibrillation as such is not transmitted to the ventricle; the ventricle, each beat of which is still co-ordinate, responds to the auricle by rapid and irregularly placed contractions (Fig. 274). When any portion of the auricle is forced into a state of fibrillation, the same condition is transmitted to all parts of it. Right and left auricle always participate, fibrillation is never confined to one or other chamber; and this is natural, seeing that, regarded as muscle, the two chambers are but parts of the same mass; the muscle is continuous, as is the masonry around and between the separate rooms of a solidly built house.

* The appearances differ according to the grade of fibrillation; I am describing an advanced grade.

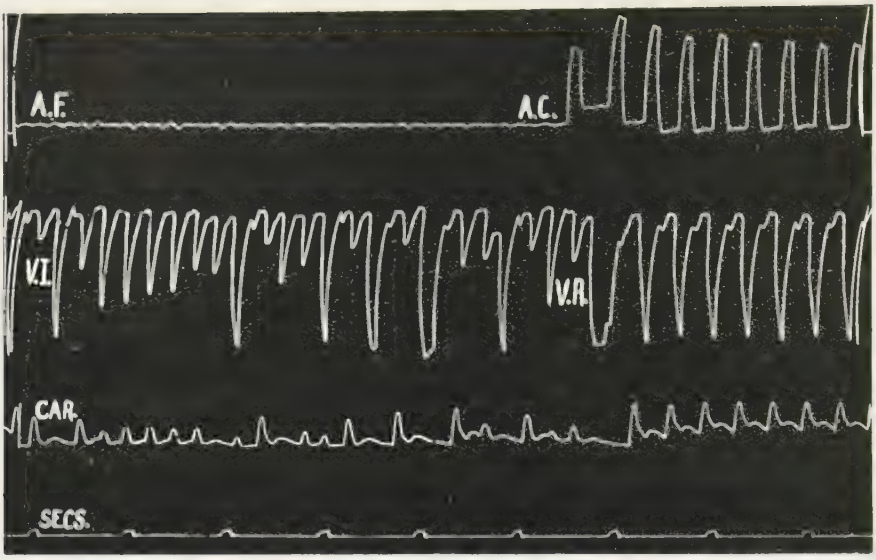


Fig. 274, ($\times \frac{1}{2}$.) Myocardiographic and carotid curves from a dog. At *A.F.* the auricle is in fibrillation, the lever stands in the diastolic position. The ventricle (*V.I.*) beats irregularly in response to the auricle. At *A.C.* the co-ordinate auricular contractions return spontaneously and the ventricular beats resume their regularity (*V.R.*). The fibrillation was induced by faradising the auricle. Time in seconds.

A curious and important feature of the auricular response to faradisation is the persistence of fibrillation after stimulation has ceased; the same phenomenon is witnessed in the case of flutter of the auricle. This "after-fibrillation" has a varying duration, and the factors which influence its persistence are still almost wholly unknown; its duration cannot be predicted, for we are ignorant of the cause which determines "after-fibrillation" or its length.

The irregular contractions of the ventricle may be studied in curves taken directly from the ventricle, or in the pulse.

Experimental and clinical arterial curves compared.

In 1899, Cushny (*86*) investigated a case of paroxysmal irregularity of the heart. He drew attention in his paper to the similarity of the arterial irregularity of the patient and that obtained when the auricles are thrown into a state of fibrillation experimentally. Seven years later he repeated his suggestion in papers written with Edmunds (*92*, *93*). Although, as he said, he did not claim to have shown a connection between the two, he suggested that the clinical condition was due to fibrillation of the auricles. The want of relation between the height of the arterial pulses and the pauses preceding them in each series of curves was remarked.

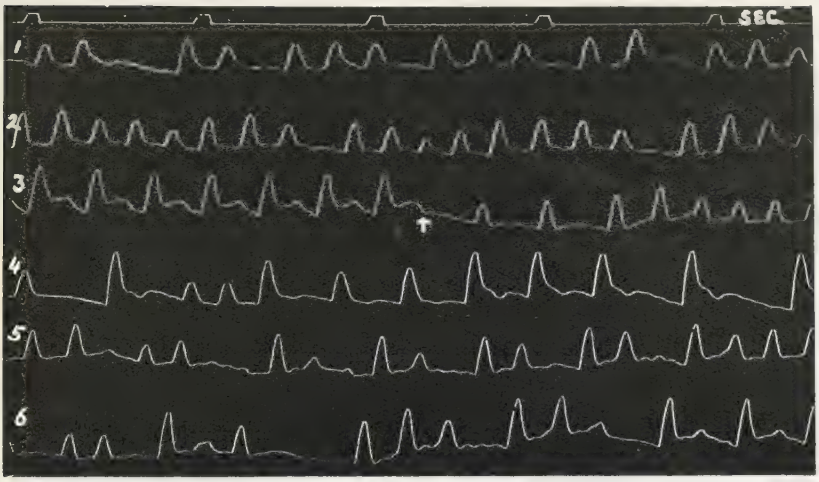


Fig. 275. ($\times \frac{2}{3}$.) (*Heart, 1909-10, I, 340, Fig 6.*) Manometric curves from the carotid of a dog, in which the auricle was fibrillating. Chest wall intact. A small portion of normal curve is shown in line 3 At the point where the arrow is placed the auricle, which had resumed its normal rhythm, was faradised and fibrillation was re-established. Time in seconds.

In so far as the experimental arterial curves are concerned, little can be added to the description given by Cushny. The irregularity of the arterial pulse in experimental auricular fibrillation is absolute and has the same qualities as those presented by the curves in complete irregularity of the heart in man. The rate of the ventricles is increased. Some examples of manometric curves taken from the carotid of dogs are given in Figs. 274 and 275. A short strip of normal curve, the only piece in the figure, is shown in the third line of Fig. 275. At the point where the arrow is placed, the auricle, which had spontaneously ceased to fibrillate, was faradised once more and fibrillation re-induced. The curves were taken with the chest wall intact and may be compared with the radial tracings given in the preceding chapter (Fig. 264, page 278).

Experimental and clinical venous curves compared.

Venous curves are easily obtained from the anæsthetised animal by means of the apparatus which is used for human work. It is only necessary to shave the hair from the root of the neck above the clavicle and to apply the receiving cup. The auricles may be thrown into fibrillation at will by means of a stimulating electrode sewn through the layers of the chest wall and into the tip of the auricular appendix. Fig. 276 opens with a series of normal heart cycles, which proceed regularly up to the point where the current was thrown in ; from this point onwards the auricles were in a state

of fibrillation and the venous curve is of the ventricular form. The auriculo-systolic wave *a* is no longer observed in the curve, each ventricular beat being represented by a pair of tall waves; later in the same curve a row of more rapid beats is accompanied by venous curves approaching the plateau type. The loss of *a* waves when the auricles are thrown into a state of fibrillation experimentally was first recorded by Fredericq, and is more clearly illustrated in Fig. 277, which is a curve also taken from a dog in which the auricles were fibrillating. In this instance the experiment had proceeded for some while and the ventricles were dilated; as a consequence the venous beats are of the plateau form.

The response of the dog's ventricle to a fibrillating auricle is usually very rapid and the diastoles are therefore short; these can be lengthened by stimulating the vagus, which slows the ventricle while the auricles continue to fibrillate. In the venous curve long diastoles often show a series of fine undulations (Fig. 278), an observation of mine (*434*) which has since been confirmed (*633*, *634*). Their presence in the experimental curves, and their production in these by the fibrillating wall of the auricle, fully explains the fine oscillations of the venous curves of man.* Briefly, the venous curves in experimental fibrillation and those associated with complete irregularity of the heart in man are alike in all respects and show similar variations. Even those human venous curves which consist almost entirely of continuous and rapid undulations and in which the ventricular elements are thereby confused (see Fig. 265*d*, page 280), are to be obtained in experiment; a fact which is illustrated by Fig. 284.

Experimental and clinical electrocardiograms compared.

Oscillations similar to those characterising the electrocardiograms in complete irregularity of the human heart are seen in experiment when the auricles are fibrillating. They were discovered in experiment independently by Rothberger and Winterberg (*660*, *662*), and by the writer (*430*, *434*) in 1909. They are invariable accompaniments of fibrillation and occur in no other disorder of the heart induced experimentally. The chief oscillations in the dog vary in frequence between 500 and 900; minute and finer oscillations which also occur are still more rapid. Though they are never quite regular in sequence, they are sometimes nearly so (Fig. 284). That they are generated in the wall of the auricle is easily proved, for the oscillations are of greatest amplitude in leads in which the contacts lie directly upon the auricular muscle; contacts placed upon the ventricle fail to show them. Direct auricular leads also show the oscillations to be continuous throughout

* According to Wiggers (*773*) these waves do not form part of the intra-auricular pressure curve as recorded by accurate instruments, but are produced, so he states with Niles in a later paper (*779*), to traction of the fibrillating auricle upon the walls of the veins. Wiggers' original conclusion that they are ventricular in origin is untenable, and the more recent conclusion that they arise by traction is one which I hesitate to accept.

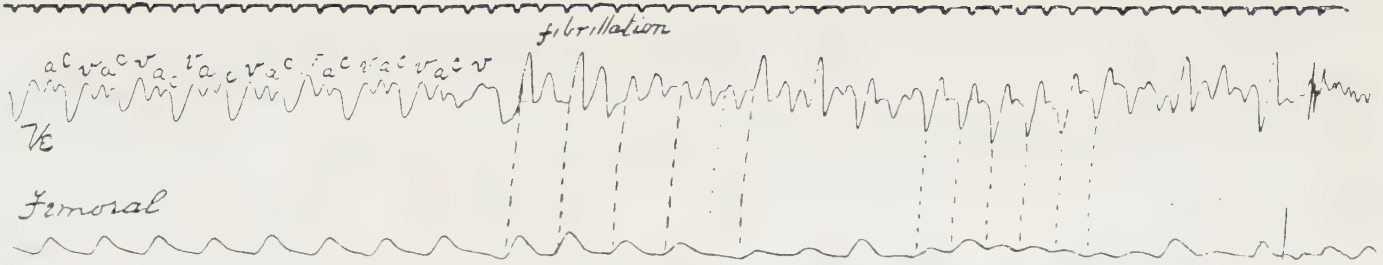


Fig. 276. (*Heart, 1909-10, I, 341, Fig. 7b.*) Polygraphic curve from a dog. Chest wall intact. The tracing commences with the normal rhythm. The auricle was faradised and the femoral pulse became irregular, while the venous pulse assumed the ventricular outline. Each pulse beat is accompanied by two prominent peaks falling during the limits of systole. There are no *a* waves. Towards the end of the tracing the ventricular beats are more rapid, and the venous pulse beats approach the plateau form.

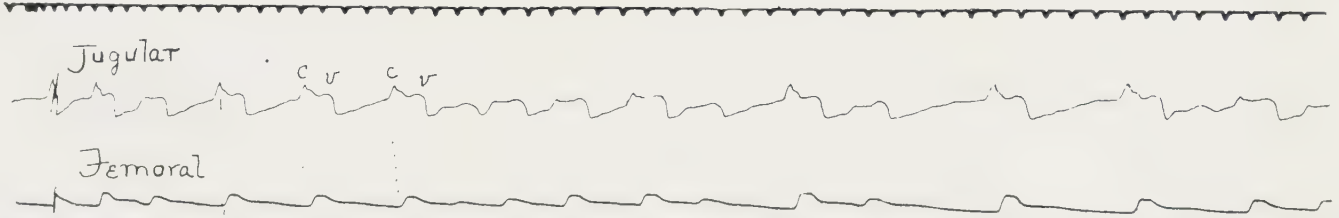


Fig. 277. Simultaneous jugular and femoral curves taken from a dog while the auricles were fibrillating. The curves were obtained towards the end of an experiment, and the heart was somewhat distended. The venous tracing is of frank ventricular form and identical with many obtained from man (compare with Fig. 267*b*, page 282).

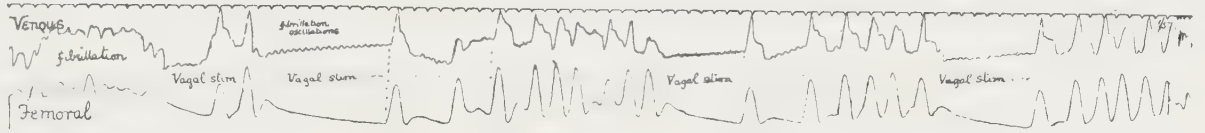


Fig. 278. (× ½.) (*Brit. med Journ., 1910, II, 1670.*) Venous and femoral curves from a dog. The auricle is fibrillating throughout, and the venous pulse is of the ventricular form. When the vagus is stimulated the ventricle ceases to beat, but the auricle continues to fibrillate, and leaves its impress on the venous curve in the form of small and rapid oscillations.

the whole cardiac cycle. That they are associated with the actual fibrillation is known, for they terminate the moment the normal heart beat is resumed. Simultaneous electrocardiographic and carotid blood pressure curves are shown in Fig. 279 and 280. In the former of these the dog's heart is acting naturally; in the second the auricles are fibrillating. During the period of fibrillation the ventricular action and the pulse are grossly irregular; the *P* summits are no longer seen but are replaced by oscillations which roughen the whole curve and distort and obscure the *T* summits. Another example of fibrillation in the dog is given in Fig. 281, in which the oscillations are particularly coarse and irregular. At times there appears to be unison between the electric oscillations and the irregular movements of a lever attached to the auricle or to the movements in the veins (Fig. 284); of curves of this kind I have published several examples, but the unison is not always maintained, and it is both rare and imperfect. If there ever is a perfect unison, it is limited to those instances in which the oscillations are very coarse and tend to be more regular, in fact to instances where there is reason to believe the curves are transitional and not those of fully developed fibrillation. Niles and Wiggers in a recent paper (*575*), in which they describe the venous curves as they are obtained with very delicate apparatus, find no correspondence between the venous undulations and the electrical oscillations: in view of these and other observations it seems probable that many of the published examples are to be attributed largely to coincidence.*

The oscillations constitute an outstanding feature of both clinical and experimental electrocardiograms. They vary considerably in form, in rate and in extent from case to case and from experiment to experiment. But when the material for selection is abundant it is possible to choose examples of curves which are pictorially alike. For purposes of pictorial comparison two curves have been selected, and are shown in Fig. 282 and 283. The former is an experimental curve of fibrillation, the second is from a clinical case of complete irregularity of the heart.

In experimental curves, as in the clinical curves, the character of the individual ventricular complex is normal. The type is repeated in all its detail whether the auricle is beating normally or is fibrillating. This is well seen in Fig. 279 and 280, in both of which *R* is notched on the upstroke. The lead adopted is a matter of indifference; the same statement is always applicable. I have examined the surface of the exposed ventricle, using a number of distinct leads; the ventricular curve remains unchanged when co-ordinate beating of the auricle gives place to fibrillation, or when the reverse change happens (*434*). In the experimental as in the clinical condition the ventricular beats are of supra-ventricular origin. Further, in experimental as in clinical curves, there is the lack of relation between the amplitude of a

* A clinical example in which approximate correspondence has been supposed to exist between coarse electric waves and vibrations on the venous curve will be found in my book (*447, Fig. 170*); the correspondence is probably never exact (*311*).

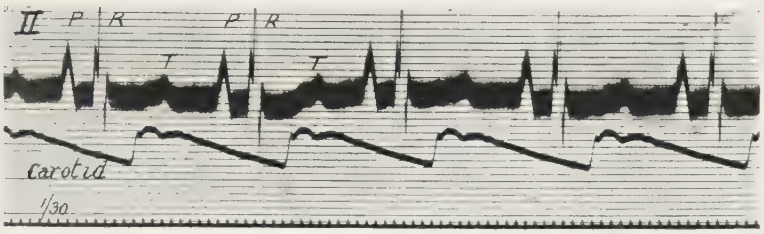


Fig. 279.

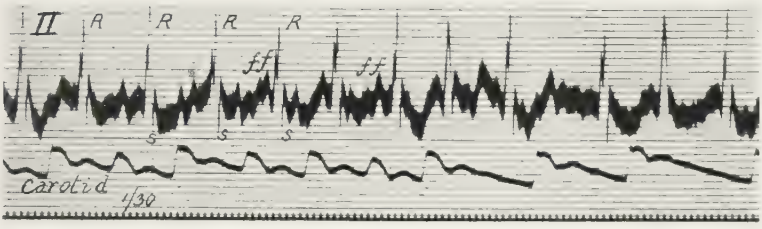


Fig. 280.

Fig. 279 and 280. ($\times \frac{9}{10}$.) Simultaneous electrocardiograms and carotid blood pressure curves from a dog. In Fig. 279 the heart is beating normally; the heart's action is regular and each ventricular contraction is preceded by an auricular beat. In Fig. 280 the auricles are fibrillating; the ventricular action is grossly irregular and rapid, the blood pressure has fallen away; the *P* summits have vanished and are replaced by coarse oscillations which render the whole curve ragged. Note the similarity of the ventricular complexes in the two curves. Time in thirtieths of a second.

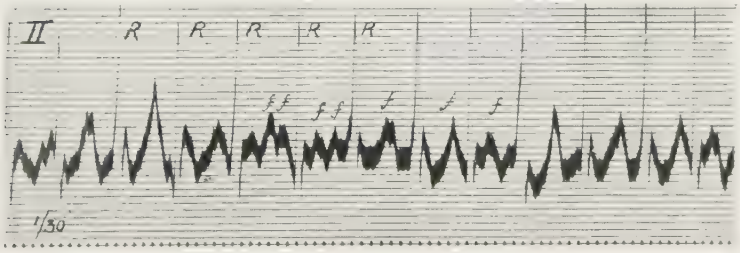


Fig. 281. ($\times \frac{9}{10}$.) Another example of a dog's electrocardiogram taken while the auricles are in a state of fibrillation. In this the chief oscillations are very coarse and irregular. Time in thirtieths of a second.

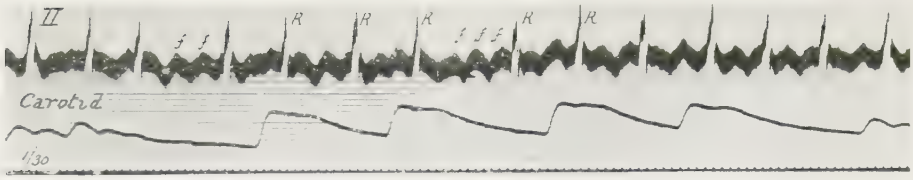


Fig. 282. ($\times \frac{3}{4}$.) Simultaneous electrocardiogram and carotid blood pressure curve from a dog in which the auricles were fibrillating. Time in thirtieths of a second.

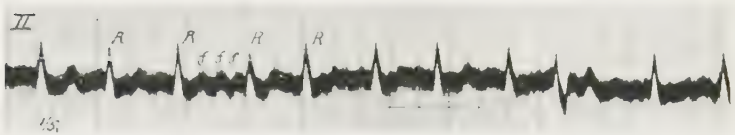


Fig. 283. ($\times \frac{3}{4}$.) An electrocardiogram from a patient in whom the pulse was completely irregular. For pictorial comparison with the last figure. Time in fifths of a second.

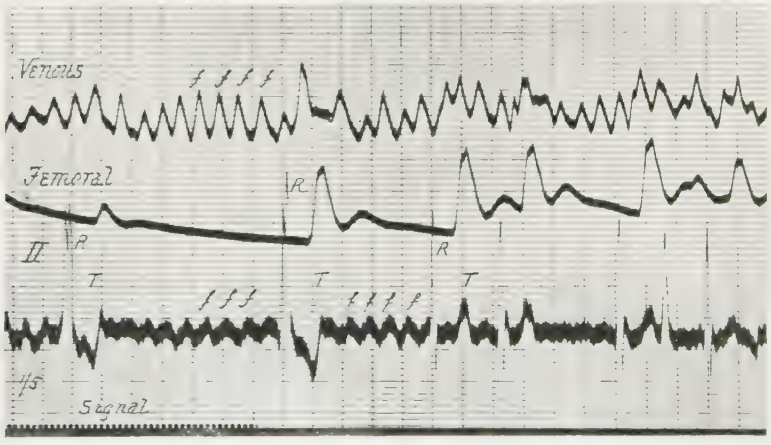


Fig. 284. ($\times \frac{5}{6}$.) Simultaneous venous, femoral and electrocardiographic curves from a dog in which the auricles were fibrillating. The signal marks stimulation of the vagus. The ventricular rate became slow during and after vagal stimulation, and during the long diastoles large undulations appeared in the veins and conspicuous oscillations in the electric curve. There is at first an apparent though imperfect unison between them, but at the end of the photograph the oscillations fade away temporarily from the electrocardiograph. Time in fifths of a second.

given *R* summit and the diastolic pause which precedes the corresponding ventricular beat, and a similar want of relation between the amplitude of *R* and the strength of the corresponding pulse beat. To some extent these phenomena are due to the clashing of *R* summits with large oscillations. If the crest of a wave coincides with *R*, it lifts it ; if the trough coincides, it depresses it ; the currents developed by auricle and ventricle are superimposed. But differences in the amplitude of *R* from cycle to cycle are often too great to be explained in this manner ; a further factor is involved which will be discussed in Chapter XXXIII.

Reviewing the electrical phenomena, it is manifest that in all their features, the clinical and experimental curves are identical. The close pictorial resemblance between selected curves from the two series, the presence of the same characteristics and of those same variations which are displayed by a detailed analysis, carries complete conviction that the clinical and experimental curves are to be ascribed to a similar disturbance of the heart's mechanism.

The harmony between the records obtained in complete irregularity of the heart in man and in experimental auricular fibrillation.

The comparison between complete irregularity of the heart in man, and auricular fibrillation in the dog is now ended. It has been demonstrated that the clinical and experimental conditions resemble each other in every respect in which they have been investigated. The observations are summarised in the following table, which consists of a systematic list of the features presented in common.

The radial curves.

1. *Rate increased as compared with the no mal.*
2. *Presence of absolute irregularity.*
3. *Absence of a constant relation between the strength of a beat and the preceding pause.*
4. *Presence of extreme variation in the force of the ventricular beats, many of which fail to reach the arteries.*

The venous curves.

1. *Presence of the ventricular form of venous pulse (the absence of "a" waves).*
2. *Presence of rapid diastolic undulations of venous volume when the heart beats slowly.*

The electrocardiograms.

1. *Presence of the supra-ventricular type of ventricular complex.*

2. *Absence of relation between the height of "R" and the length of the preceding diastole. Absence of relation between the height of "R" and of the corresponding pulse excursion.*

3. *Absence of "P" summits.*

4. *Presence of characteristic oscillations which are shown to be generated in the auricular portion of the heart. The persistence of these oscillations throughout the whole cardiac cycle.*

Although auricular fibrillation is now universally recognised as the cause of complete irregularity of the ventricle in man, and although no one who has read and understood the evidence now doubts this conclusion, there was a time not long ago when many hesitated to accept it.

The disinclination to believe in what was at first hypothesis is attributable to the frequence with which the human heart is affected by this disorder, by the tradition, now exploded, that irregularity of the heart is the result of distension of its walls, and by the occasional persistence of the irregularity for many years. It seemed inconceivable to those imbued with the mechanical theory of heart disease that so profound a disturbance of the auricular function could persist for many years. A case had been recorded (*349*) in which the irregularity had lasted for five and a half years and Mackenzie had watched cases unchanged for even double this period. Recently indeed, a case in which the disorder had in all probability persisted for 32 years has been placed on record (*249*). At the time of which I am speaking (1909-1910) the hypothesis was received with scepticism and it was necessary to collect and record all the available evidence. The proof as I gave it was written much as I have written it in these two chapters and was to my mind conclusive. But there still remained those who doubted, and it was for this reason that I attempted to obtain a further evidence which would silence all criticism. I turned to the lower animals in the hope of discovering the fibrillation of the auricles as a manifestation of disease in them. After a while I was fortunate in finding what I desired.

Auricular fibrillation in the horse.

As a very rare affection, complete irregularity of the heart is manifested by horses, who are the subjects of cardiac disease (*434*, *450*). In these animals, as in man, it is often associated with breathlessness, rapid exhaustion and other signs of distress, and, as in man, it may be accompanied by enlargement of the veins, by ascites and by general dropsy. The ventricular rate

is increased from the normal of 30 or 40 per minute up to 60 or 70 or even to as much as 150 per minute. The disorderly action is characteristic in that the lengths of the cycles are quite lacking in uniformity. I have had the opportunity of examining six horses with this disorder of the heart. Curves obtained from the arteries and veins are indistinguishable from others obtained from human patients; the venous curve shows ventricular waves, *c* and *v*; the diastoles are devoid of *a* waves, but from time to time display the small undulations which are so typical of the condition. I have also obtained electrocardiograms from one animal; and in these the usual features were to be observed. The irregular ventricular complexes were all of the supra-ventricular type, *P* summits were absent, oscillations were present, though ill-defined.

In one of these animals I was able through the kindness of General F. Smith and Colonel Blenkinsop to make the desired observations. The animal, which presented the characteristic symptomatology and a gross irregularity of the ventricular beat, was thrown and shot through the brain. The right side of the chest was quickly opened and an excellent view of the heart was obtained, the ventricle continuing to beat rapidly and irregularly. At first no intrinsic movement could be seen in the auricle, its walls seemed fixed in diastole, but close inspection revealed the fibrillary movements. The epicardium covering the right auricle is sufficiently thick to be somewhat opaque in the horse and fine movements in the underlying muscle are not easy to see. But the independent movements of the light reflections in the little surface ridges, especially over the appendix, were perfectly clear and were demonstrated to and recognised by Colonel Blenkinsop and others who were present during these observations.

Thus came the final and ocular proof that experimental fibrillation as we know it, occurs as a manifestation of disease.

Much of the evidence in these chapters may now seem redundant; sufficient evidence to carry conviction might be set forth in a few paragraphs. My decision to preserve these chapters in a form similar to that in which they appeared in "The Mechanism of the Heart Beat" has come not only from a belief that, where a conclusion of first consequence is at stake, the reasons for that conclusion should be set forth fully, but also in the hope that these chapters may serve to illustrate how persistent study may build up and consolidate our knowledge of phenomena at first obscure—obscure because they manifest themselves in the deep seated and hidden tissues of the human body.

CHAPTER XXV.

AURICULAR FIBRILLATION, HEART-BLOCK AND EXTRASYSTOLES.

Fibrillation and heart-block.

In the healthy heart fibrillation of the auricles drives the ventricles at a greatly enhanced rate. In experiments in which the heart is in a fresh condition the ventricular rate rises to double or even treble the normal. But under some experimental, and under many clinical, conditions the ventricular rate may be normal or may even be reduced notably. Thus, in patients the ventricular rate may lie anywhere between 30 and 200 per minute ; the rate of the ventricle is controlled exclusively by the state of the conducting tissues, so far as we know at the present time. That the impulses of fibrillation are transmitted through the *A-V* bundle, as are the normal impulses, was first shown by Frederieq (*187*). He found that, when the bundle is broken, the auricle though continuing to fibrillate no longer affects the ventricle. His experiment I have often repeated and substantiated. Damage of the *A-V* bundle, conduction being abolished completely or partially, has similar effects while the auricles are fibrillating and while they are beating at an accelerated rate. When the function of this bundle is suppressed altogether, the ventricle develops its own regular rhythm. When its capacity to conduct is merely impaired, the rate of ventricular response is lowered, and the degree to which the rate is lowered goes hand in hand with the degree of damage. But the rate at which successive impulses impinge upon the junctional tissues in fibrillation favours the display of block, as does a simple acceleration of the auricle. A slight impairment of conduction is sufficient materially to reduce the ventricular rate.

If we examine all the known ways of reducing the ventricular rate while the auricles fibrillate, we shall find that heart-block may always be ascribed as the cause. The same cause is alone found to reduce the ventricular rate in clinical cases. The chief facts are set forth under the succeeding subheadings.

Complete dissociation and auricular fibrillation.—Transection of the *A-V* bundle while the auricles fibrillate is followed by standstill of the ventricle, and subsequently a slow and regular idio-ventricular rhythm is

developed. If complete heart-block is first induced, either by section or by asphyxiation, and the auricles are then caused to fibrillate, the regular ventricular rhythm persists unaltered.

When I first suggested that slow ventricular action in clinical cases of fibrillation is the result of heart-block (*431, 434, 479*), I cited a notable patient. A syphilitic subject, he suffered from the Adams-Stokes syndrome. The ventricular action ranged constantly about 30 beats per minute and was usually regular (Fig. 285). From time to time ventricular extrasystoles were seen (Fig. 286) and these cycles were of the same lengths as the regular cycles. During his fits the ventricle ceased to beat. The ventricle behaved in every respect as it does in simple and complete heart-block. Yet the picture

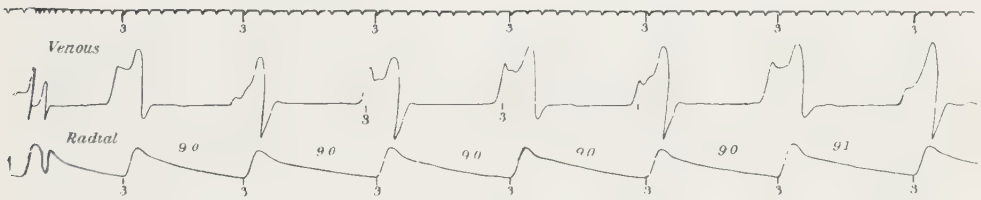


Fig. 285. ($\times \frac{5}{7}$.) (*Quart Journ. Med., 1909-10, III, 273, Fig. 1.*) Polygraphic curve from a case of complete heart-block and auricular fibrillation. The venous pulse is of the ventricular form; the radial pulse is slow and regular.

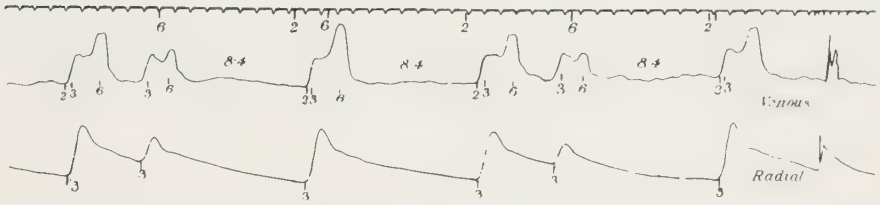


Fig. 286. ($\times \frac{4}{7}$.) (*Quart. Journ. Med., 1909-10, III, 273, Fig. 2.*) Polygraphic curve from the same case as Fig. 285. The ventricle is beating irregularly because ventricular extrasystoles disturb its rhythm. The returning cycles are of the same length as the initial cycles.

differed from simple dissociation in that the venous pulse (Fig. 285) was of the ventricular form, showing no sign of auricular contractions. In electrocardiograms no *P* summits were to be discovered, but replacing them were the usual oscillations in characteristic form. The heart, obtained at a later date (*73*), exhibited a complete break in the *A-V* bundle by an old-standing syphilitic lesion. A similar example has more recently been reported with post-mortem evidence (*192*). Other cases* have been collected and my

* Though lesions of the *A-V* system have naturally not always been discovered (*338*).

original conclusion that heart-block is responsible for a slow ventricular response, when fibrillation is associated with a slow action of the ventricle, has now received a wide measure of support and confirmation (*169*, *192*, *221*, *350*, *368*).

In some patients complete heart-block is induced by heavy doses of digitalis (Chapter XIV). I have known the ventricle to become slow and perfectly regular under the influence of this drug in more than one case of fibrillation (*106*, *451*, *516*, *717*). In such instances the ventricular rate is relatively high,* usually from 40 to 50 per minute, but occasionally as high as 90 per minute (Fig. 287).

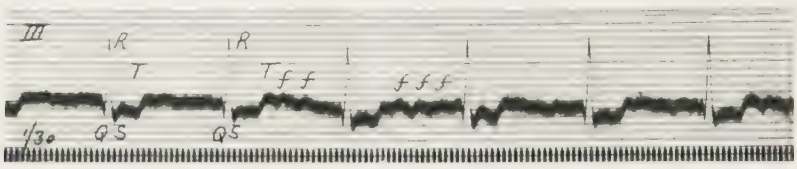


Fig. 287. (*Heart*, *1911-12*, *III*, *279*, *Fig. 12*.) From a case of auricular fibrillation, under treatment with digitalis. The fibrillation is evidenced by the oscillations *f*, *f* and by the absence of *P* summits. The ventricular action is regular because complete heart-block is present. The rate is exceptional for a ventricle in complete heart-block, being approximately 90 per minute. Time in thirtieths of a second.

Partial heart-block and auricular fibrillation.—A simple method of producing partial heart-block of different grades in experiment, is by asphyxiation. If a cat is asphyxiated, while the auricles fibrillate, the ventricular responses are soon reduced in number, and continue gradually to be reduced if asphyxia is maintained. The rate of the ventricle is governed by the degree of block (*439*).

Amongst clinical cases there is an interesting group. The patients to whom I refer present heart-block and later develop fibrillation of the auricles. I have drawn attention (*434*) to a description under the term "nodal bradycardia" of such a case by Mackenzie (*516*); and other examples have since been published (*169*, *228*). In all these patients fibrillation has been associated with a slow ventricular action.

The post-mortem evidence in cases of slow ventricular action is not of itself convincing. Many hearts from patients with slow and irregular ventricular action have been examined, and lesions of the *A-V* bundle of varying severity have been found (*56*, *103*, *170*, *192*); but in respect of these it cannot be said, any more than it can be said of straightforward partial block, that the extent of the visible lesion goes hand in hand with the degree of slowing; neither is such a precise relation to be expected.

* Presumably because the block occurs at a high level of the *A-V* node, and because the ventricle is governed by a rhythm formed in the *A-V* node.

The effect of vagal stimulation upon auricular fibrillation.—Stimulation of the vagus, while the experimental auricle is fibrillating has one of two chief effects (*397*, *597*, *647*, *650*). If the fibrillation is of short standing, it may suppress it. If it is of longer duration, it usually fails to do so. The inhibitory impulses are accompanied by a certain alteration in the auricular mechanism itself, the movements in the walls of the auricle appear to be more finely subdivided, suggesting the establishment of lines of block in the musculature. But the most conspicuous effect is slowing of the ventricle (Fig. 288 and 289). Perfect clinical examples of this retardation have been published by Wenckebach (*763*). He has shown that firm pressure upon the

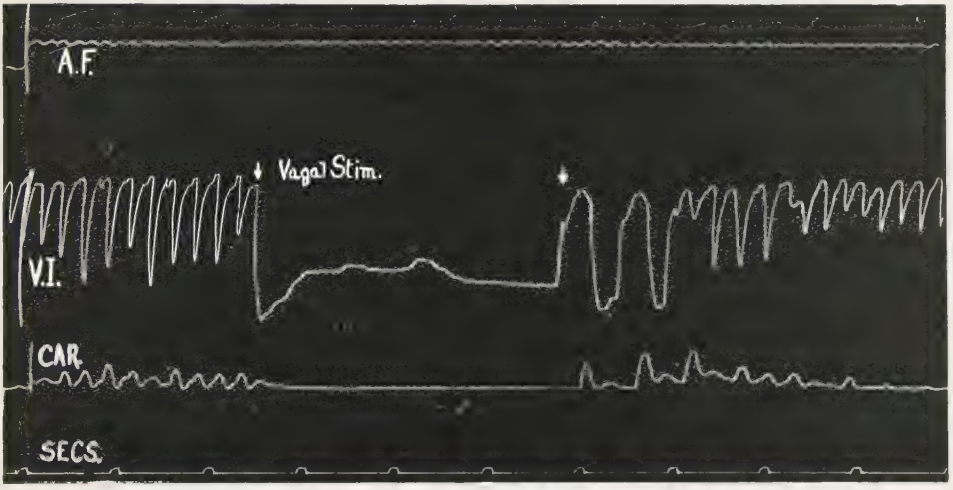


Fig. 288. ($\times \frac{1}{2}$.) Myocardiographic curves (from auricle and ventricle) and carotid pressure curve from a dog. *A. F.*, the auricular curve, while the auricle is fibrillating. *V. I.*, the ventricular curve, showing the irregularity of the response to the fibrillating auricle, the standstill of the ventricle produced by vagal stimulation and the subsequent recovery. Time in seconds.

carotid sheath may cause conspicuous slowing of the ventricle in man when the auricles are fibrillating (Fig. 290). This observation has been repeatedly confirmed (*447* (*Fig. 187*) *and 653*).

The manner in which the rate is reduced is not in doubt, for if in an experiment the normal auricular action is resumed before the vagal effect has subsided, simple heart-block is seen (*434*). The vagal effects illustrate the rule that a slow ventricular rate, when the auricle is fibrillating, may be ascribed to decreased conduction power of the *A-V* tissues. The final illustration is that provided by digitalis and its allies.

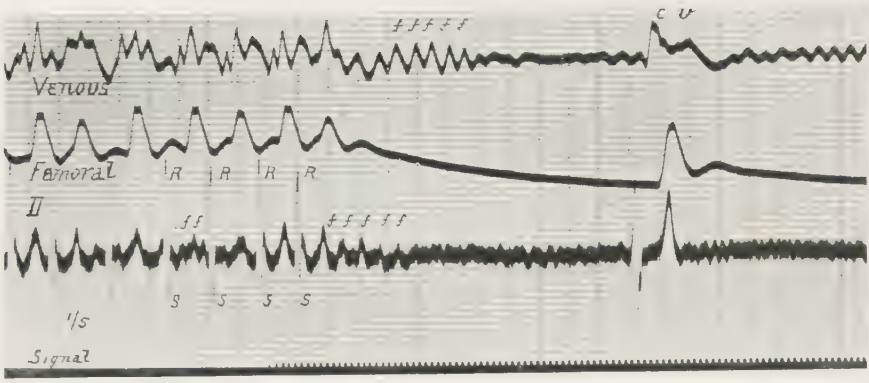


Fig. 289 ($\times \frac{4}{5}$.) Venous, femoral and electrocardiographic curves from a dog, during the progress of auricular fibrillation. The vagal stimulation is indicated by the signal. The ventricular action, at first rapid, is conspicuously reduced; the oscillations in the veins and in the electric curve are reduced in size. Time in fifths of a second.

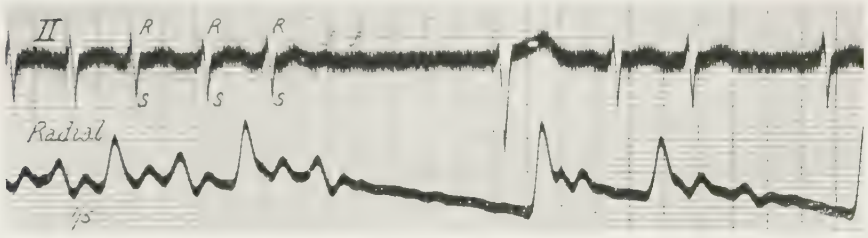


Fig. 290. ($\times \frac{6}{7}$) Arterial and electrocardiographic curves from a patient. Fibrillation of the auricles is present. The ventricular action, at first rapid, has been conspicuously reduced by pressing upon the carotid sheath. The vagus was compressed at about the period indicated by the third time line. Time in fifths of a second.

Digitalis in auricular fibrillation.—The potency of digitalis in slowing the ventricular pulsations in the condition now termed auricular fibrillation was first demonstrated by Mackenzie (*507*, *508*), and has since been confirmed abundantly (*95*, *106*, *168*, *517*, *700*, *771*). The action of this drug in this respect, so I have concluded, is to be ascribed to heart-block (*431*, *439*); this conclusion first suggested itself to me because I was aware that digitalis induces heart-block when the rhythm is normal. It is in fibrillation that the pace of the ventricle may often be set with nicety by regulating the dose of the drug. In these circumstances the ventricle slows gradually, but eventually profoundly (Fig. 291), while it continues to beat irregularly in most cases. Exceptionally, as has been pointed out, complete block may develop and the ventricle then beats regularly.

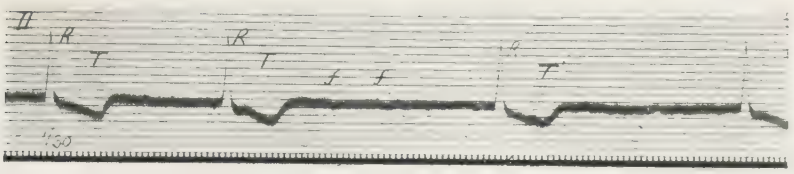


Fig. 291. Fibrillation of the auricles from a patient under treatment with digitalis; the ventricular action is very slow. Time in thirtieths of a second.

My conclusion that digitalis produces heart-block in fibrillation and that the slowing is due to this cause is now generally accepted. But the exact manner in which this slowing is brought about has been much discussed. In a few patients atropinisation of the heart, which has been retarded by digitalis, produces little or no acceleration; in these cases there is presumptive evidence that the vagi are not responsible for the slow rate. In most patients the slow heart action is accelerated, often conspicuously, by atropine(*95, 517, 700*). Thus, in these patients there is little doubt that the actual slow rate under digitalis is in part due to vagal tone; but whether as an effect of digitalis this tone is *greater than the normal vagal tone* is debated. To be certain that there is such an effect, it has to be shown that the rise of ventricular rate following atropine is to *the same level* when atropine is administered before and after the heart is brought under digitalis; in which case the whole of the digitalis slowing might be attributed to the vagus. This rise to a given level in the two circumstances has only been observed in exceptional cases; as a prevailing rule, the rate reached under atropine in the non-digitalised heart is greater, usually much greater, than that reached by the atropinised and digitalised heart. In such cases, as Cushny (*91, 95*) has rightly pointed out, a conclusion that the slowing is vagal is not warranted.

Cushny (*91*) emphasises the direct action of digitalis, which he believes to be peculiar to hearts affected by fibrillation; but that is not strictly the case, for some cases of simple heart-block appear to be unrelieved by atropine. The direct action of digitalis seems to be absent when the heart is quite normal, as in experimental animals; in these digitalis slowing is abolished by section of the vagi or by atropinisation.* Cushny shows that, if the perfused heart is used, digitalis exerts also a direct influence upon the bundle, and concludes that malnutrition is responsible for this effect in experiment and also in certain instances of disease. It is difficult to believe that an action on the vagi is not at least in part responsible for the actual drop of the ventricular rate when digitalis is employed in auricular fibrillation; for the

* Halsey (*239*) has shown that in dogs under digitalis or strophanthus full doses of atropine or amyl nitrite will abolish the block. Curiously, small doses of either drug will *produce* a measure of block, whether the heart has been submitted to digitalis previously or not. This is but one example of the now well-known reversed action of atropine in small and large doses.

powerful slowing effect of the vagi in this disorder is well-recognised, and digitalis is known to increase vagal tone in other patients and in experiment. If digitalis does not in part slow the ventricle through the vagi, but only by a direct action on the bundle, then it is necessary to assume that the pharmacological effect of digitalis upon the vagus is absent in cases which present this form of disordered heart action. Provisionally, therefore, it seems safer to conclude that the digitalis effect is twofold, that it acts in part directly, in part indirectly, and that its relative power to act through one or other channel is variable in different patients. At the same time, a review of all observations emphasises the direct action.

Similar slowing is obtained with the allied drugs, strophanthus (*5*) and squills (*124*, *735*, *788*); both drugs produce heart-block, but whether through the vagus or directly is still uncertain.

The known causes of ventricular slowing may be summed up by repeating that they are always such as induce heart-block. Naturally there are clinical cases in which heart-block cannot be directly proved. For instance, in those in whom the auricles are always fibrillating and the ventricular action is constantly slow; but in these also we are justified in at present assuming that heart-block is responsible for the low ventricular rate.

Fibrillation and ventricular extrasystoles.

According to current hypothesis fibrillating auricles shower impulses upon the ventricle indiscriminately; it is in this way that the complete irregularity of the ventricular response is explained. The ventricular cycles are of very variable lengths. If it could be shown that many cycles are of equal length—so many that their equality could not be ascribed to coincidence then either the hypothesis would become untenable or some complicating factor would have to be proved.

Now we have seen that while the auricles are fibrillating, the ventricular action may be perfectly regular; in this circumstance, the rate is slow because heart-block is present. But ventricular cycles of constant length sometimes appear in another form. The condition has been termed, somewhat loosely, "digitalis coupling." When the auricles are fibrillating and the heart comes fully under the influence of digitalis, the ventricular action is retarded. It is as an accompaniment of this retardation that coupling is usually seen (*507*). The irregularity is singular and distinctive (Fig. 292). The beats of the ventricle occur in pairs and the short cycles are almost or quite constant in length. Variation is still seen in the long cycles; this is to be expected and is consistent with our view of fibrillation, for the ventricular systoles which terminate the long cycles, and govern their lengths, may be attributed to haphazard auricular impulses. Not so the beats which terminate the short cycles (*i.e.*, the second beats of the pairs), for the pauses which precede these are of uniform duration. If our hypothesis is sound,

these beats must be of different origin. Electrocardiograms show them to be so as I was able to demonstrate (*434*, *436*) some years ago (see also *107*). Fibrillation of the auricle is shown in Fig. 293 ; the majority of the ventricular complexes conform to the supraventricular type. But the curve opens with a period of " coupling " ; the second beat of each couple has the shape of an extrasystole arising in the left ventricle. That such is the origin of these anomalous systoles is, I think, beyond doubt, for digitalis in full doses is known to provoke extrasystoles. The pairing of the beats is similar to the

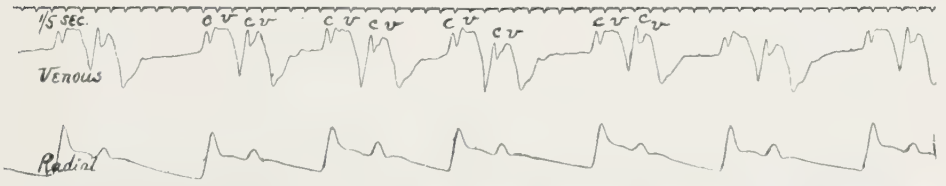


Fig. 292. Venous and radial curves showing so-called " digitalis coupling " The arterial beats are in pairs ; the interval between the first and second beat of the couple is uniform ; the pauses following the pairs of beats are variable in length. The venous curve is of the ventricular form.

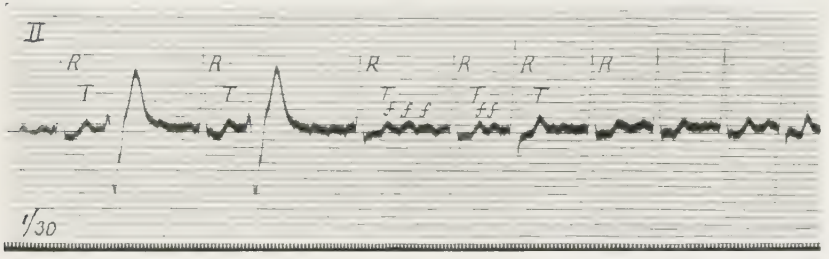


Fig. 293. An electrocardiogram showing a period of ventricular coupling ; later the ventricular complexes are quite irregularly placed and are then all of the supraventricular type. Time in thirtieths of a second.

pairing which manifests itself when extrasystoles disturb a normal rhythm. The reason why the short cycles during the period of coupling are not variable in length is that the second beat of the pair is not a response to an auricular impulse.*

Coupling of the form described may appear independently of digitalis, but whether it is the result of overdosage with this drug (Fig. 294) or whether it occurs under conditions less clearly defined, the electro-

* Very occasionally the ventricular complex is constant in form throughout a period of " coupling " ; this fact is not out of harmony with my conclusion ; in such cases the second beats of the pairs may be regarded as extrasystoles arising in the junctional tissues. The cause of " coupling " is further discussed in Chapter XXVIII.

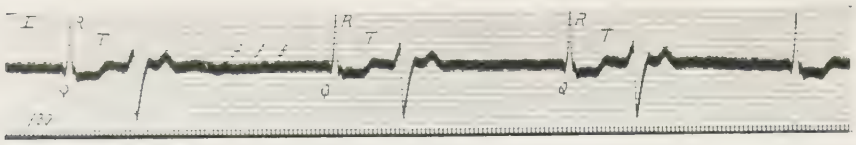


Fig. 294. ($\times \frac{3}{4}$.) From a case of auricular fibrillation on large doses of digitalis. It shows a coupling of ventricular beats which speaks of over-dosage. The first complex in each couple is of the supraventricular type; the second is of different form; these last contractions are premature and originate in the ventricle. Time in thirtieths of a second.

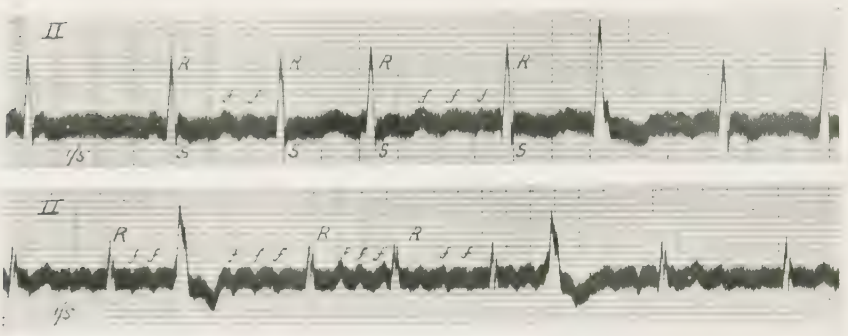


Fig. 295. ($\times \frac{9}{10}$.) Two clinical curves showing fibrillation of the auricles. The responses of the ventricle are for the most part to auricular impulses; but from time to time an isolated extrasystole appears; such extrasystoles arise in the ventricle; it will be noticed that they fall early in the preceding diastole. Time in thirtieths of a second.

cardiograms present the same pictures. Single extrasystoles frequently complicate auricular fibrillation (*275*, *434*, *436*) (Fig. 295); the same beats may appear in short runs. In some patients all the extrasystoles are from one focus; in other patients they may be derived from several sources. The forms of extrasystoles in a given patient are constant from month to month. In patients who suffer from them, if fibrillation is subsequently acquired, the extrasystolic irregularity persists; the anomalous beats may be shown in these circumstances to be of the same outline before and after fibrillation is acquired (*441*), an observation which places their extrasystolic origin during the period of fibrillation beyond reasonable doubt.

Chapter XXVI.

VENTRICULAR FIBRILLATION.

It was found by Hoffa and Ludwig (*320*), in 1850, that the normal beat of the ventricle is abolished by applying to this chamber strong constant or faradic currents. The ventricle so assaulted exhibits very rapid irregular movements of minute amplitude; co-ordinate contraction of the fibres seems to be lost; the heart at first shrinks, but soon swells and no longer expels its contents. The condition outlasts stimulation and usually persists until all movement in the heart ceases.

The phenomena presented by the fibrillating ventricle have been described on many occasions and are familiar to numerous workers (*520*). During the final stage, when the ventricle is ballooned and the arterial pressure has for long sunk to zero, the whole wall of the chamber is convulsed by minute quiverings; over the whole superficies twitchings of the muscle are visible. If attention is concentrated upon a small area, little waves of contraction may be perceived to pass along the muscle bands for a short distance, the course of the wave altering from moment to moment, but always dying out abruptly and often at a given line. It seems from inspection alone that while one small area is contracting, the adjacent area may be in a state of relaxation. At the same time a more general movement is often perceived; it is as though a ring of tissue surrounding the cavities contracted more firmly, and as if this ring of contraction travelled from apex towards the base or base towards the apex. Yet the inconstancy of the picture from moment to moment perplexes the observer and the intricacy of the contractions and relaxations defies analysis. This fully developed condition is properly referred to as one of *ventricular fibrillation*.

Fibrillation may be induced, not only by the application of electric currents, but by other irritants, thermal, chemical and mechanical. Especially is this the case when the ventricle is in an irritable state; according to McWilliam (*522*), "weak faradic currents, a touch with a hot wire, a mere scratch with a pin . . . or even slight pressure with the finger, are each sufficient at such times to excite the fibrillar contraction." Spontaneous fibrillation, by which I mean the onset of fibrillation with no apparent cause,

not infrequently terminates experiments on the heart. Fibrillation usually follows the sudden occlusion of a coronary artery or of a main branch; a fact known for a long while to many (*78*, *609*).

The injection of large doses of digitalis (*85*), potassium chloride (*522*, *546*), and many other toxic bodies will cause it.

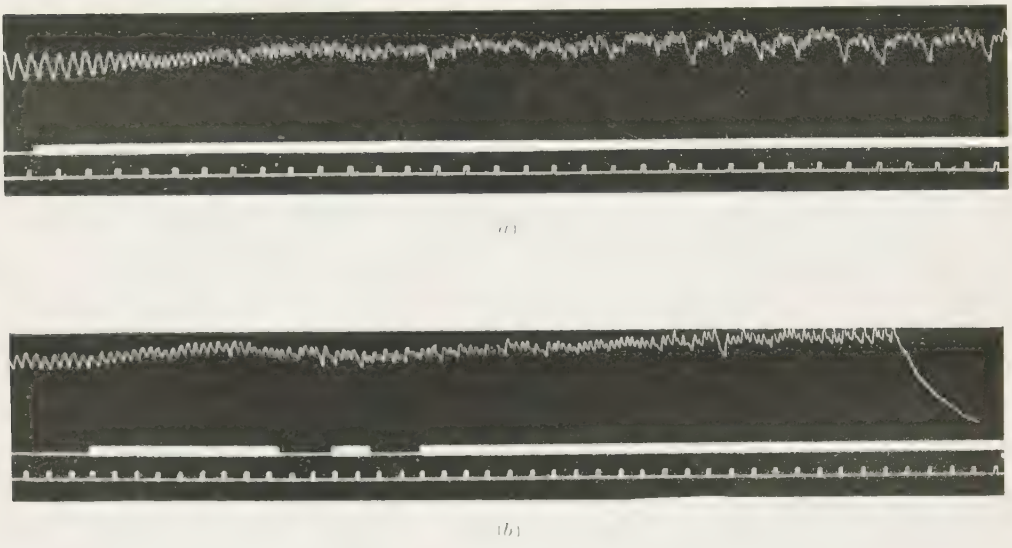


Fig. 296. (*Levy. Heart, 1912-13, IV, 335, Fig. 7.*) Carotid curves of the carotid pulse in a cat. The first curve shows the effect of stimulating the right stellate ganglion of the sympathetic while the animal was under the influence of 2 per cent. chloroform vapour. The normal rhythm is replaced by an irregular tachycardia. The second curve shows the effect of stimulation under 0·5 per cent. vapour. A similar tachycardia is produced but it terminates in fibrillation of the ventricles, the blood pressure falling to zero. The white bands are the signals of stimulation. Time in seconds.

Still more notable, because of their direct practical bearing, are the experiments of Levy (*421-423*, *425*, *426*). Working upon cats, he has shown that the heart is extremely susceptible to low percentages of chloroform vapour inhaled, and that when it is under the influence of this drug, ventricular irritability is enhanced to a precarious degree. The injection of minute doses of adrenalin, stimulation of the sympathetic (Fig. 296),* section of

* The relation of ventricular fibrillation to nervous impulses is a subject which it will be convenient to defer to Chapter XXIX.

the vagi, and stimulation of a sensory nerve, are each capable of forcing the sensitised ventricle into fibrillation; and when the conditions are favourable, interferences, so inconspicuous as to escape observation,* may precipitate the state.

That the fibrillation of the ventricle is not due to destruction or paralysis of a co-ordinating centre, as Kronecker and Schmey (*396*) imagined† has been forcibly argued by McWilliam (*522*) and, in the light of present knowledge, this hypothesis has been almost universally abandoned (*407*). The whole excised ventricle, or any considerable part of it which is cut away, may be made to beat co-ordinately or in a fibrillating fashion; fibrillation is primarily due, as McWilliam states, to changes within the ventricles themselves and is not caused, when induced by faradisation, by abnormal impulses transmitted through the nerves from other parts or by direct stimulation of surface nerves; the state of fibrillation is conveyed from one part of the ventricle to another and will cross the bridges between zig-zag incisions which penetrate the walls. Transmission from one part to another is determined solely by there being sufficient muscular union (*211*, *459*). Outstanding features of the condition are the complexity and persistency of the movement. McWilliam concludes that it is related to the complex arrangement of the muscular fibres in the ventricular walls, for the complexity of movement is greater in the heart of adults than it is in the young, it is fully developed in the mammal, whereas in cold-blooded animals the response to faradic stimulation is far less complex in nature (*24*, *407*). Garrey (*211*) expresses the relation in more simple, and probably more correct, terms; he believes that the proclivity to fibrillation is related to the mass of tissue involved, because he finds it cannot be induced in small pieces of ventricular tissue and because the smaller the mass of tissue the less persistent is the condition. All observers are agreed that recovery may take place sometimes; it occurs occasionally in the dog, frequently in the cat, and it is the rule in the rat and mouse (*237*, *522*); the quivering movement ceases and after a brief quiescence, comparable to the pause‡ following a paroxysm of tachycardia (*792*), the heart will beat again in a normal fashion. Recovery is distinctly controlled by the size of the organ and possibly, therefore, by the degree of the original derangement. It will be convenient to defer a discussion of the ultimate nature of fibrillation to a succeeding chapter, in which this and several closely allied problems may be treated together, and meanwhile to describe the records of fibrillation and to note the events which lead up to that state.

* Indirect stimulation such as happens when a fresh dose of stronger vapour is inhaled may be cited; as may also the simple act of lifting the animal.

† Kronecker and Schmey came to this conclusion when they saw fibrillation follow the introduction (often repeated introduction) of a needle into the ventricular septum at a given level. The same result is often to be obtained when other regions of the ventricle are similarly treated and by many less violent procedures.

‡ The pause is shorter than is the compensatory pause because impulses from the fibrillating ventricle are transmitted back to the auricle.

Records of fibrillation.

When fibrillation of the ventricle is induced by faradic stimulation, gradually increasing in strength or maintained; when it is brought about by ligation of a coronary artery (*432*), by the administration of chloroform (*426*) or by the injection of digitalis: or when it is precipitated by nerve stimulation or the injection of adrenalin, the essential events are the same. Fibrillation in its fully developed form does not come abruptly; it is foreshadowed by a distinct train of events. The premonitory period may be long or short; on occasion the steps are incomplete, but when complete they are definitely ordered. At first the heart's regularity is disturbed by premature contractions

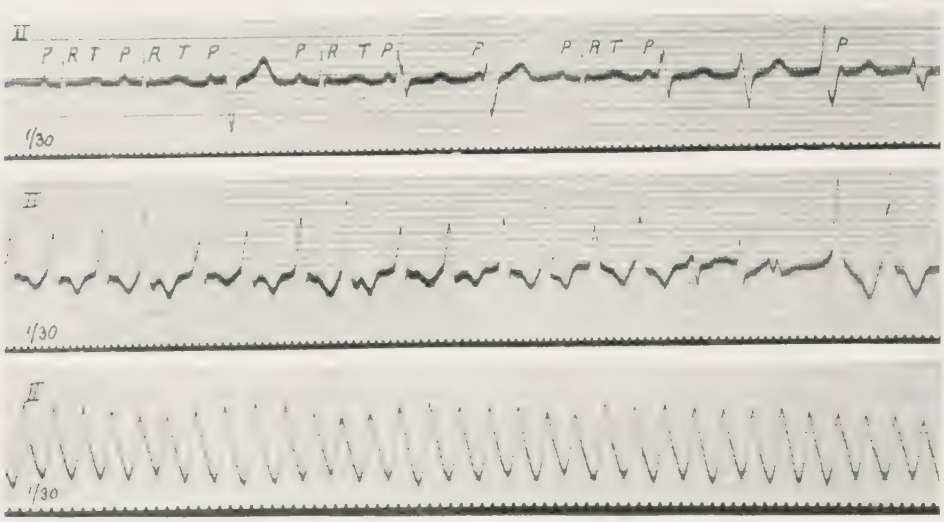


Fig. 297. Three electrocardiograms, illustrating the disorders produced in the cat's heart by the inhalation of low percentages of chloroform vapour. The first curve shows the normal rhythm, interrupted by ventricular extrasystoles arising in at least three separate foci. The second curve shows the fully developed tachycardia; the beats are not uniform in shape, their origin is variable, neither are they quite regular in incidence. The third curve exhibits the mechanism which immediately precedes fully developed fibrillation of the ventricles. This disorder probably resembles closely what is termed flutter in the case of the auricle. Time in thirtieths of a second.

coming from a ventricular focus; these increase in number and eventually form groups; other foci of activity are often added (Fig. 297). The heart accelerates in response to these new impulses and is eventually controlled by them exclusively: a tachycardia originating in a single ventricular focus, or in several ventricular foci, becomes established.

It may be gradually, it may be more suddenly, that the rate of movement increases till the circulation can be maintained no longer; the arterial pressure rapidly sinks to zero and the process develops further. At this

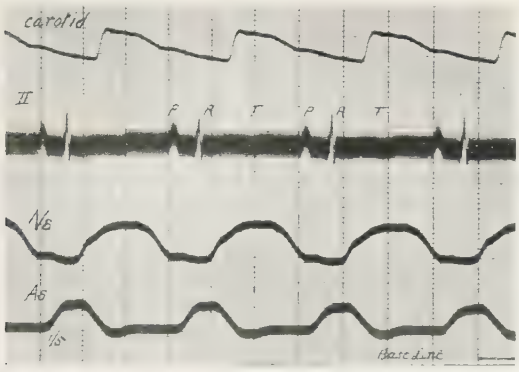


Fig. 298.

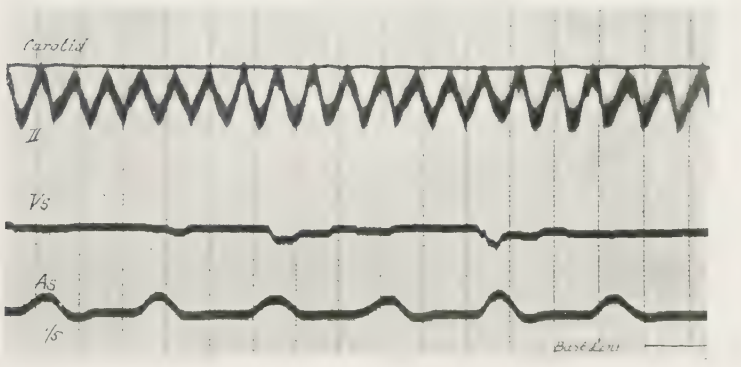


Fig. 299.

Fig. 298 and 299. ($\times\frac{2}{3}$.) Simultaneous carotid blood pressure curve, electrocardiogram and ventricular (*Vs*) and auricular (*As*) myocardiograms from a dog. Fig. 298 was taken before and Fig. 299 shortly after faradising the ventricle; the second curves show the early stage of ventricular fibrillation. In Fig. 299, the electrocardiogram displays large oscillations, following each other at a rate of 375 per minute. The excursions are not quite uniform in shape or sequence. The blood pressure (the carotid curve may be measured to the common base line of the photographs) has fallen almost to zero, though little undulations still show upon it. The auricle (*As*) continues to beat, its rate being enhanced; its beats are not regular. The ventricular myocardiogram shows only a few coarse movements, which correspond to the changes in the muscle; this lever lies in a position of not quite full systole. Time in fifths of a second.

stage the movement witnessed in the ventricular wall is a rapid undulation, almost regular, but of small amplitude. The ventricle is not dilated but, on the contrary, is diminished in volume. If stimulation is withdrawn at this stage the action continues for a little while and ends in recovery, or progresses as it would if stimulation were continued. The electrocardiogram

shows a movement of the fibre exceeding in amplitude that of the natural ventricular beat ; the individual undulations (Figs. 297, last curve, and 299) are almost regular in incidence, but often varying in amplitude in a phasic way.

The auricle beats with increased frequency and irregularity (*208*, *792*) ; it is responding to the ventricle, for its movement becomes regular if the bundle is divided (*76*). The movements of the ventricle are scarcely strong enough to be recorded, but a few general or jerky movements may be seen (Fig. 299). The further change to the fully developed

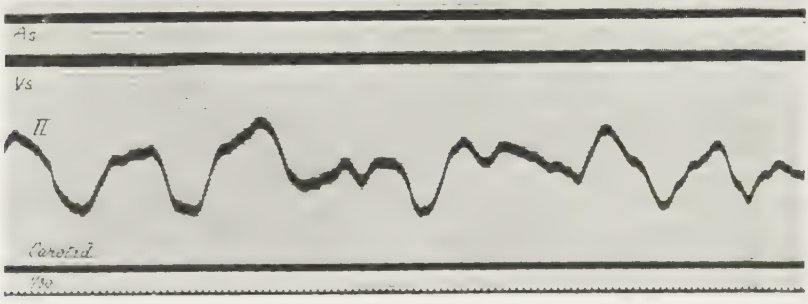


Fig. 300. (× ⅔.) Similar curves from another animal and showing the final and full picture of ventricular fibrillation. The levers recording movements of auricular and ventricular muscle and carotid blood pressure are motionless. The electrocardiogram consists of very large and irregular oscillations. This type of curve corresponds to the phase of ventricular dilatation when small flickering movements are visible on the surface. Time in thirtieths of a second.

condition, which in my view alone deserves the term fibrillation (see Chapter XXVIII), is so gradual that its onset cannot be signalled. The heart gradually distends and all movements of the auricular and ventricular levers cease (Fig. 300) ; the blood pressure remains at zero ; the appearance of the ventricular surface is that described in the first paragraph of this chapter ; the movements of the galvanometric string assume the characteristic irregularity (*358*) illustrated by Fig. 300.

Clinical fibrillation.

It is to be remarked that all the major forms of cardiac disturbance which have been produced experimentally are also known to occur clinically. Until recently a single condition, fibrillation of the ventricle, formed an exception to this statement. If fibrillation of the auricles is so frequent in the human subject, it is natural to ask why a similar disturbance of the ventricle is so rare ? Its rarity is presumably more apparent than real ; for fibrillation of the ventricles spells death.* We have now the strongest

* This is the rule in the dog : recovery is rare ; it is almost certainly the rule in man, though an exception has been recorded by Robinson and Bredeck (*652*).

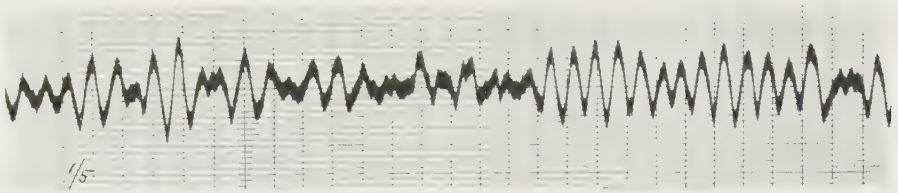


Fig. 301. ($\times \frac{3}{4}$.) An electrocardiogram showing the irregular and rapid oscillation of the string, when in ventricular fibrillation the ventricle has recently dilated. This curve represents a transition between those of Fig. 299 and 300. Time in fifths of a second.

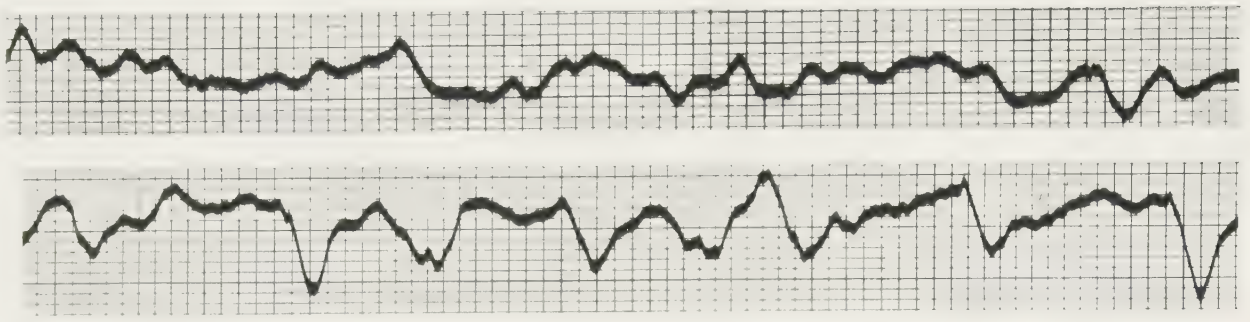


Fig. 302. (*Halsey. Heart, 1915, VI, 67, Fig. 10 and 11.*) Examples of ventricular fibrillation, occurring in a patient at the time of death. Time in fifths of a second.

a priori reasons for believing that sudden and unexpected death comes to many patients in a manner suggested by McWilliam in 1889 (*524*). That unexpected death may follow embolism or other obstruction of a coronary artery has long been recognised (*23*, *250*, *656*); experiment indicates that fibrillation of the ventricle is the immediate cause of these catastrophes. Unexpected death is not infrequent in patients who suffer from fibrillation of the auricles, especially when the heart is fully reacting to digitalis. Ventricular fibrillation as the cause of such death is suggested not only by the known presence of a similar auricular disorder and by the known irritant effects of digitalis upon the ventricle, producing in these patients ventricular extrasystoles and in animals fibrillation as a culminating reaction, but also by the manner of death. The suddenness of the event, the immediate loss of all signs of heart beat, while pallor gives place to a deepening cyanosis, and the few gasping respirations which contribute the last signs of life, all point in the one direction (*297*, *434*, *451*). Levy's conclusion, originally suggested by McWilliam,* that ventricular fibrillation is a cause of death under chloroform, probably the only cause of any moment (Levy), is upheld by a convincing array of facts and arguments (*422*, *425*, *526*).

Clinical records.—There are but few examples of curves from the human subject purporting to show fibrillation of the ventricles; the attendant circumstances are alone responsible for the scarcity of such records. The first published electrocardiogram is that of Hoffman (*327*, *328*); it is not acceptable as an instance of fibrillation, but rather of a transitional stage, a rapid tachycardia springing from several ventricular foci. It occasioned a complete loss of the pulse for some time, but the patient recovered. That the ventricle was on the verge of fibrillating cannot be doubted. The action of the heart in patients dying of anterior poliomyelitis was recorded by Robinson (*646*). In one of two curves published, fibrillation is depicted. Electrocardiograms were taken by Halsey (*238*) from a patient dying of broncho-pneumonia; the actual death of this patient coincided with fully developed fibrillation of the ventricles, which in this instance is unmistakably shown by the curves (Fig. 302). Robinson and Bredeck (*652*) have recently placed another instance on record; during one of several attacks of syncope, curves of fibrillation were obtained; the patient recovered temporarily and curves showing auricular and ventricular systoles in sequence were then photographed; death occurred thirty hours after the recorded attack. In the electrocardiograms the characteristic oscillations are seen to be rapid and relatively uniform; the attack, as judged from these records, was not one of fully developed fibrillation but of the last of those preliminary disturbances which precede full fibrillation. The curves of Robinson and Bredeck represent a less advanced grade of disorder than do those of Halsey.

* McWilliam saw fibrillation occur when excessive doses of chloroform were given and concludes that it is not a primary cause of death in healthy animals (see *525* and his "Report on an experimental investigation of the action of chloroform and ether." *Brit. Med. Journ.*, *Oct. 1890*). The importance of Levy's work lies in his demonstration that light anæsthesia is dangerous.

Chapter XXVII.

The Heterogenetic Impulse, and the Inter-relation of Extrasystole, Paroxysmal Tachycardia and Fibrillation.

In past chapters I have dealt for the most part with matters of fact and with such conclusions as have received general acceptance.

But it is impossible for any worker who has devoted many years to the study of disordered heart action to avoid speculation, or to be free from general conceptions which may or may not justify themselves in the end. While I hold it to be the duty of a writer on matters scientific to exercise restraint in putting forward views to which he is not wedded, yet certain of those conceptions which have conspicuously and successfully guided his own observations may profitably be disclosed. In placing certain views on record in this and the immediately succeeding chapters, I do so with reserve; whereas the facts as they are at present known seem to me to uphold them, I am aware that they must remain *sub judice*, and that knowledge acquired in the future may render them untenable.

The hypothesis of heterogenetic contractions.

The first hypothesis which I propose to discuss is one to which I have long adhered (*447*), namely, that the heart is capable of originating contractions of two vitally different types, that its beats may be of the physiological or homogenetic order or of the pathological or heterogenetic* order. The proof that there is a vital basis for this distinction I am unable to give; the evidences which suggest it will be enumerated. I emphasise this view,

* In again using this term I desire to avoid further misinterpretation or confusion of my meaning. The term heterogenetic I use to apply to the beat which arises in the same way as an extrasystole. The term refers to the process of generation and in no sense to the locality in which the beat arises; a beat arising in an abnormal *place* I designate as *ectopic*. Now a homogenetic beat *may* be (though usually it is not) of ectopic origin, for instance the beats which belong to an *A-V* rhythm; and the heterogenetic beat may not be (though usually it is) ectopic, for instance the so-called sinus extrasystole. I use the term heterogenetic in much the same sense as Hering has used the term *myoerethic* (*253*).

believing that, so long as it may reasonably be held, we are not justified in assuming that what has been shown in the case of one order of beat may be assumed for heart beats generally; failure to distinguish has confused more than one outstanding problem.

To introduce the conception, I contrast two phenomena, already considered in detail, namely, the ventricular extrasystole and an escaped beat of the ventricle; these may be taken to exemplify the heterogenetic and homogenetic types, respectively. The most remarkable character of the extrasystole is its prematurity; it occurs long before a beat of the heart is expected. I am aware that extrasystoles may occur so late in diastole that they fall almost at the instant when the next systole of the ventricle, in which they arise, is due; but such exceptions are immaterial to the argument; the extrasystole of the ventricle frequently follows the end of the previous refractory period by time intervals of no more than a twentieth, or even a fiftieth of a second. In contrast with this is the escaped beat of the ventricle; it is the rule that such beats terminate long diastoles; long periods of rest always precede them. In simple instances of ventricular escape, escape may be predicted; the ventricle escapes whenever the diastole is of sufficient length. It is like the bursting of a boiler when pressure is rising; it is inevitable when the pressure comes to a certain level; but that rise of pressure takes time, there is an appreciable preparatory period. The bursting of the boiler is deferred, indefinitely may be, when a valvular escape anticipates the critical pressure; close the valve and after a period has elapsed the explosion comes. The opening of the valve lowers the pressure, which re-accumulates as the valve closes. In the ventricle, contraction takes the place of the valve; escape of the ventricle is deferred by each systole of the chamber, which, so we infer, nullifies the preparatory process. The extrasystole comes without warning, it comes after this contraction of the ventricle and not after that. Whatever the preparatory process may be, it is not continuous and inflexible. The process which leads to ventricular escape has the quality which underlies rhythmicity; the process which determines the extrasystole has not this quality. An extrasystole is followed by a pause during which there is abundant time for its repetition, yet it is not repeated. It may be repeated, it is true, but repetition is the exception and not the rule; were it the rule, extrasystoles would never stand isolated.

Treat the question on a wider basis and include, as homogenetic, beats of the normal rhythm or those of an independently beating ventricle; include, as heterogenetic, those of paroxysms of tachycardia. The two orders maintain their apparent distinctions. The paroxysms are rapid, the rhythm of the *S-A* or *A-V* node is relatively slow; here is the same essential difference as between the ventricular extrasystole and the ventricular escape. When a series of beats is generated in the muscle of the heart, this series is ordered at its beginning in one of two ways. On the one hand there is a gradual acceleration, a quickening until the final rate is assumed (Gaskell's rhythm of development), that is a rhythm of physiological or homogenetic type. On

the other hand there is an abrupt onset, the heart rate springing at once to its full rate; that is a rhythm of pathological or heterogenetic type. The environment which favours the one does not favour the other; the growth of physiological impulses is favoured by normal nutrition; the extrasystole, the paroxysm of tachycardia, is favoured by abnormal nutrition or poisoning. Influences which depress homogenetic impulses, for example the injection of potassium chloride, often excite heterogenetic beats.

Another striking difference between rhythms of the two kinds is to be found in the nature of their nerve control. The physiological rhythm of

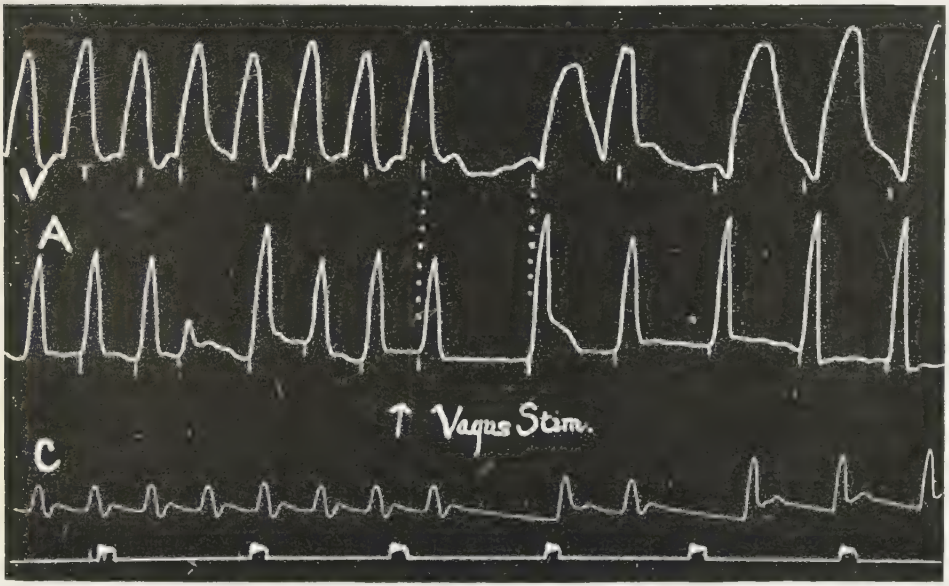


Fig. 303. (*Heart 1909-10, I, 116, Fig. 10.*) Myocardiographic curves (*V* = ventricle, *A* = auricle) and carotid pressure curve (*C*) from a dog. Over the first half of the curve a tachycardia arising in the ventricle is present and the auricle is responding to it (as shown by the relation of the contractions and by the single premature auricular contraction which disturbs the auricular rhythm only). Vagal stimulation inhibits the paroxysm. Upon stimulating the vagus the new rhythm ceases abruptly and the normal rhythm is resumed. A single reversed beat, of a similar nature to the paroxysmal beats, interrupts the restored *S-A* rhythm. Time in seconds.

the *S-A* or *A-V* node is under the graded influence of the vagus; stimulation of the nerve slows the rate, more or less, according to the strength of stimulation. Paroxysms of tachycardia are under a precarious control,*

* Although they may arise from the same focus as the homogenetic rhythm (*i.e.*, those of *A-V* nodal origin).

the *rate* is not altered on stimulating the vagus, either there is no effect or the new rhythm abruptly ends and the normal rhythm is resumed. This fact has been established (*432*) in respect of paroxysms excited in the ventricle by occluding a coronary artery experimentally (Fig. 303), and in respect of clinical paroxysms of tachycardia (*31*, *64*, *649*).

The chief centres of physiological impulse formation in the heart are known ; they are the *S-A* node and the *A-V* node, the bundle and perhaps its chief branches play minor rôles.* Extrasystoles definitely known to arise in either of these two nodes are comparatively rare.† The birth of homogenetic impulses is especially, though not exclusively, associated with tissue of special architecture. The physiological rhythm is more rapid when propagated from tissue lying nearest to the superior cava, and as we pass to other centres the rhythmic properties are found to be less highly developed. The rates of pathological rhythms are in no degree distinctive of the region of the heart from which they come.

The reasons are many and strong which warn us never to speak or think of all impulses generated by the heart as being of one kind, or as being influenced in the same manner by conditions of environment. It is imperative that the two types of which I here speak should be considered and studied separately.

The heterogenetic impulse as the prime factor in many disorders of the heart's action.

The second hypothesis, which I propose to consider and which it has been necessary to assume to a limited extent in the preceding paragraphs, is that the heterogenetic impulse is a prime causative factor in a number of closely allied disorders of the heart's action (*447*). In addition to the extrasystole, paroxysms of tachycardia, flutter and fibrillation are ascribed primarily to impulses of this kind. There are many evidences that these four conditions are very closely allied. The simplest example is the relation of extrasystole and paroxysmal tachycardia. Patients who present such paroxysms customarily exhibit isolated extrasystolic disturbances of the normal rhythm during periods of relative quiescence ; it is the rule that these extrasystoles emanate from precisely the same region of the heart as do the

* In the frog's heart, all parts of the muscle are capable of originating rhythmic impulses ; many parts of the mammalian heart may also be capable of doing so in special circumstances (*147*).

† Yet, it should be observed, it is not possible to declare positively that they ever arise elsewhere than in the special tissues. The auricular extrasystole showing an inverted complex is thought by some to arise in the upper regions of the *A-V* node ; other extrasystoles in which the auricular complex is anomalous, but not invert, may arise from the lower stretches of the *S-A* node. Extrasystoles are generally considered to arise in the *A-V* node at different levels. There is little to show that ventricular extrasystoles can arise from ventricular muscle as opposed to Purkinje tissue.

paroxysms themselves, not only from the same heart chamber, but, as the electrocardiogram tells us, from the same part of that chamber. In other cases transitions from one form of disturbance to the other are witnessed; the heart's action is now disturbed by an isolated extrasystole, now by a pair of premature beats (*585*), now by a run of three or more following closely upon each other, eventually by a group of sufficient number to constitute what is unhesitatingly termed a paroxysm. Such observations inevitably suggest that the paroxysm consists of repeated extrasystoles. The disturbing beats all originate in the same focus (*428, 433*); the isolated extrasystoles and the first beat of a paroxysm have the same degree of prematurity; the isolated extrasystole and the paroxysm is followed by a pause of the same kind before the normal rhythm is resumed. Even more significant is the similarity between those interferences which induce extrasystoles and paroxysm (see Chapter XXIX), and the similarity of interferences which abolish these disturbances. In experiment almost any interference which will produce the paroxysm will, if acting in diminished force, produce the extrasystole. Influences which cut short the paroxysm, for example, flushing the heart with blood by pressing on the abdominal veins (Fig. 304), will also abolish the extrasystole. The conclusion is inevitable, and I think now almost universally adopted, that the *paroxysm is composed of a series of extrasystoles.* The question why extrasystoles are repeated may be deferred for the moment.

The relation of extrasystole to flutter and fibrillation is more difficult to establish, from the circumstances of the case; yet there is a good deal of direct evidence and still more indirect evidence to support it. The quiescent periods which alternate with attacks of flutter and fibrillation in man are customarily disturbed by extrasystoles. The clinical attacks are longer and the events which lead up to them and immediately follow them have not been fully unveiled. The abrupt onset and cessation are suggestive. In the only patients in whom the beginning and ending of brief attacks of auricular flutter have been recorded (*644, 645*) the events have been similar to those seen in simple paroxysms.* In experimental flutter of the auricle the disturbance ends in a pause in a degree comparable to that following an extrasystole (see Fig. 249, page 264, and Fig. 250, page 265). In experimental fibrillation the onset is preceded by extrasystoles. The experimental procedures which are known to induce flutter or fibrillation, are also known to induce extrasystoles.

Many clinical observations are also upon record, which point to the close relation of simple paroxysms of tachycardia and fibrillation. Such for example, are those described by Hewlett (*309, 310*) and by myself (*434, 487*),

* Semerau (*698, Fig. 2*) publishes a curious curve in which he considers that a short attack of flutter is shown. It is difficult to argue from this curve, in that the oscillations are very rapid and are not regularly placed. It is possibly transitional between flutter and fibrillation for it exhibits very coarse waves.

in which rapid and regular tachycardias of auricular origin have given place, more or less abruptly, to fibrillation of the auricles. In the only curves I have seen (*698*) in which the onsets and endings of attacks of fibrillation are clearly shown in detail the events are inconstant. One paroxysm of unmistakable fibrillation begins in an auricular extrasystole; in another patient fully developed paroxysms start quite abruptly. The author also refers to paroxysms foreshadowed by tachycardia of auricular or atrio-ventricular origin. In Semerau's patient the paroxysms of fibrillation

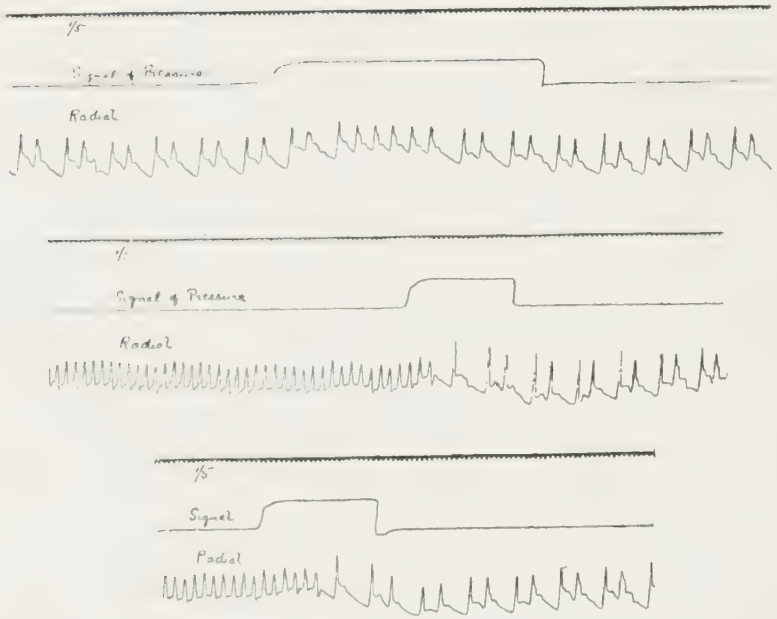


Fig. 304. ($\times \frac{2}{3}$.) Three curves showing the effect of firm pressure on the abdomen in a patient who displayed disorderly action of the heart. In the first tracing the regular action is interrupted by auricular extrasystoles occurring after each second natural cycle. These extrasystoles are temporarily abolished by pressure on the abdomen, and a row of six natural cycles occurs. In the two lower tracings long continued paroxysms of tachycardia of auricular origin are cut short by a similar procedure.

end quite abruptly, the oscillations showing no material preliminary change, though in one curve they become finer before the fibrillation ceases. The length of pause between the last oscillation and the first normal auricular beat is unhappily not measurable.

The facility with which auricular flutter in patients may be converted into fibrillation is now widely recognised, and this knowledge has assumed therapeutic importance. In patients who exhibit auricular flutter (as described in Chapter XXII) digitalis slows the ventricle by increasing the pre-existing heart-block, the auricle meanwhile continuing its original

action. But if the drug is pressed, the flutter in the auricle gives place to fibrillation. This phenomenon, which I was first able to demonstrate in a patient (*451*) treated by Mackenzie, I have repeatedly confirmed (*457*) as have many other observers (*336*, *635*, *644*). It is a reaction which can often be obtained in these cases. Therapeutically it is of importance in that if the drug is withdrawn when fibrillation is established the auricular action often changes again, reverting to a normal action and not to flutter (Fig. 305). Thus, it would seem as though the original exciting cause of flutter is not continuous in its action, but that flutter once established

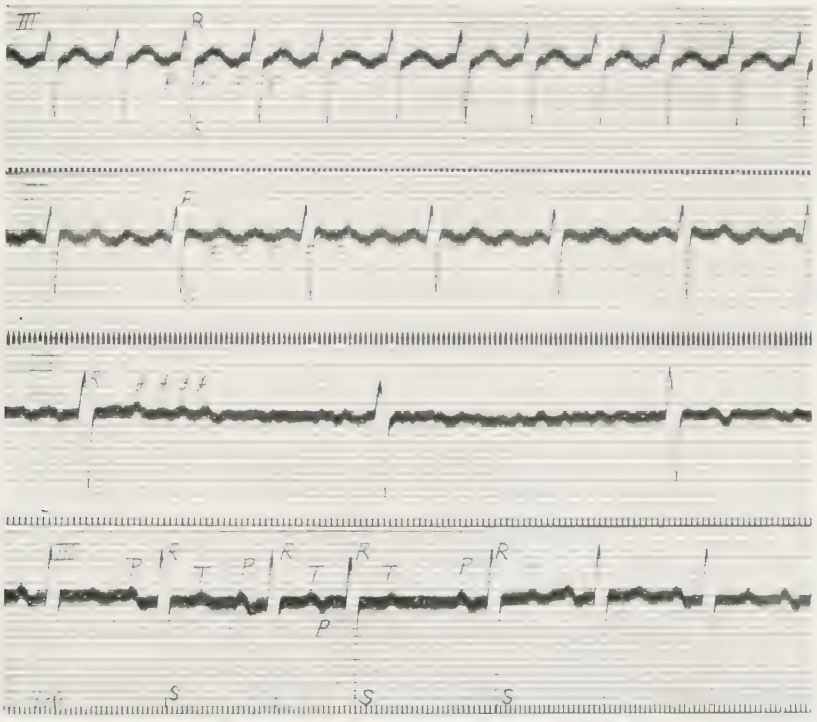



Fig. 305. Four curves from a case of auricular flutter; showing the effects of treatment. The first curve shows an auricular rate of 300 and a ventricular rate of 150. In the second curve the auricular rate is maintained, but the ventricular rate has been halved (4 : 1 block is present) as a result of digitalis administration. In the third curve auricular fibrillation is seen and it is accompanied by a slow and irregular action of the ventricle. In the last curve the normal rhythm, interrupted by occasional premature contractions of auricular origin, has been resumed.

tends to maintain itself. Patients who suffer from auricular flutter, may develop fibrillation in the absence of digitalis medication, a fact which further emphasises the relation between the two conditions. In experiment

also I have seen a similar transition following upon stimulation of the vagus. Fig. 306 exemplifies this statement; flutter in the auricle had been induced by weak faradic excitation of its wall; the auricle was beating regularly at a rate of 500 per minute, the ventricle meanwhile responding at half this rate. Stimulation of the vagus, while the heart continued to beat in this fashion, immediately threw the auricles into a state of fibrillation. Several clinical curves have been published purporting to show the transition from fibrillation to flutter (*644*, *645*), but they do not show it satisfactorily. There is, however, a curve of Parkinson and Mathias's (*589*, *Fig. 4*), which, I believe, exemplifies this change, though it is not so interpreted by the authors. A possible example of the reverse action, the passage of fibrillation to flutter under vagus stimulation, is shown in Fig. 307. In its opening phases this curve shows the irregular oscillations and an irregular action of the ventricle. During stimulation the auricular waves become almost, if not quite, regular (*f*, *f*) and develop a constant and distinctive form, in which the summit shows notching.

A paper recently published by Rothberger and Winterberg (*674*) emphasises the close relation of flutter and fibrillation. Indeed, these writers go so far as to state that coarse-waved fibrillation and flutter as observed in man are identical, and that flutter and fibrillation generally are but different grades of one and the same condition. As the auricle recovers from faradic stimulation its movements often become gradually coarser and reduce in rate up to the moment when the normal rhythm is resumed. It is with this coarse "fibrillation" that Rothberger and Winterberg deal; they term it flutter. They state that during this phase of the heart's action a relation exists between electrical oscillations recorded directly from the auricle and the *ventricular responses*.

Such periods of cardiac disorder as yield these coarse waves and verge on "fibrillation" on the one hand and "flutter" on the other have given rise to much speculation. One of the earliest analyses of such a curve will be found in my paper on auricular fibrillation (*434*, *Fig. 29*). The mechanism was considered at that time to be transitional between fibrillation and the normal rhythm; an imperfect 2 : 1 ratio is shown between auricular oscillations* and ventricular beats. Rothberger and Winterberg's studies are upon similar curves and their object is to show that the transitional curves seen in these circumstances are in a measure analysible.† Nevertheless, the view that the majority of these curves are pure "flutter" curves and identical with those seen in so-called flutter in the human subject, is one

* Taken, not from a direct lead, but from lead *II*.

† Robinson (*648*) supposes that this coarse-waved condition is a mixture of fibrillation and flutter. In his view the term fibrillation should be confined to the fine-waved variety, a definition which has much to recommend it. But with a theory of mixture which supposes the simultaneous pressure of the two disorders in the same muscle area I do not find myself in agreement, any more than do Rothberger and Winterberg.

with which, for several reasons, I do not concur. The oscillations yielded by the auricular tissue in the experiments follow each other at a rate of approximately 500 per minute, but the deflections are *not* sufficiently regular in their incidence in the published curves. A feature of flutter in man is the remarkable regularity of the auricular movements; as stated in Chapter XXII, exact measurement fails to find an average variation of more than 0·008 seconds. In the experimental curves which we are discussing the variation is much greater; that "flutter," as it is understood in the human subject, forms the basis of the disorder may possibly be true, but that most of these curves are those of simple and uncomplicated flutter as seen in man can scarcely be allowed. The curves are transitional curves and are very possibly produced by impulses arising in a relatively few auricular centres.* In some of Rothberger's curves this is definitely suggested by variation in the forms of the separate oscillations.† If further evidence were required that such curves are transitional and not purely "flutter" it would be found in the spacing of the ventricular responses. According to the writers themselves it is only when simple ratios are established that analyses are possible, a good proportion of their analyses are unconvincing; in human "flutter," where the ventricle is beating irregularly, an analysis is always possible‡ and demonstrates remarkably precise time-relations between auricular and ventricular contractions, however irregularly the latter may be disposed (see Chapter XXII).

To return to the general question, namely, the relation of extrasystole, forms of paroxysmal tachycardia and fibrillation, there are on the experimental side further significant illustrations. If stimulating electrodes are placed on the auricle and a very weak faradic current is thrown into the muscle, the first disorder of the heart's action which results is the solitary extrasystole; increase the strength of the current and groups of these beats appear; increase it further and the heart is thrown into rapid and rhythmic action; again strengthen the current and this regular tachycardia becomes confused and passes by insensible gradations to fibrillation. When the auricle is contracting at a high speed in response to external stimulation,

* The irregularity, slight though it is, by itself suggests this.

† In other curves the form is almost, if not quite, uniform, but this is not to be interpreted in the sense that the impulses are unifocal in origin, seeing that but a minute part of the auricular tissue was under investigation.

‡ Amongst a very large number of pulse curves from flutter cases in which the ventricles beat always or from time to time irregularly, I have seen no instance in which a perfect analysis of the responses could not be made for the great majority of the ventricular cycles; I have only seen two instances (out of some thirty cases) in which this analysis could not be completed for *every* ventricular cycle recorded. In the two cases referred to, the analysis remained in doubt only here and there. These analyses are possible in clinical flutter where 2 : 1, 3 : 1, 4 : 1, 5 : 1, 6 : 1 and 8 : 1 ratios are intimately mixed, if sufficient time and attention is given to the curves. They are possible because the auricular rate is so constant from moment to moment and the *As-As* intervals so unvarying.

or in response to new though intrinsic impulses, it is ever on the verge of fibrillating. Precisely similar events are seen in the ventricle, and can be induced in orderly sequence in this chamber by various procedures, such as electrical stimulation, ligature of a single coronary artery (*432*), or poisoning with small percentages of chloroform (*426*). Whether it be in the auricle

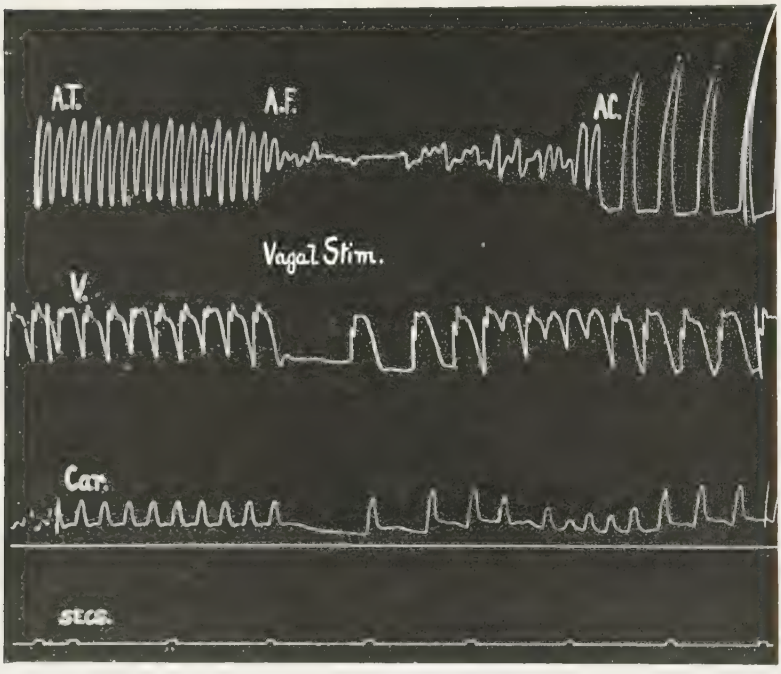


Fig. 306. Myocardiographic curves (A = auricle, and V = ventricle) and carotid pressure curve from a dog. At $A.T.$ the auricle is fluttering in response to a weak faradic stimulation, the ventricle is beating at half the auricular rate. At $A.F.$ the auricle passes into fibrillation as a result of vagal stimulation. At $A.C.$ the co-ordinate action of the whole heart is resumed. To illustrate the close relation of flutter and fibrillation. Time in seconds.

or in the ventricle, whether it be caused by this interference or by that, the natural sequence of events is as follows: 1, extrasystole; 2, groups of extrasystoles; 3, simple or complex tachycardia or flutter; 4, fibrillation. It may be said further that unless very powerful stimuli are suddenly applied to the heart (auricle or ventricle) it cannot be thrown directly into fibrillation without the interposition of premonitory states. It is true that the series is not always complete and that one or more of the steps

may be omitted; but preliminary disturbances, leading up to the final delirium are almost if not quite constant in occurrence. We are led to conclude that the process which underlies the extrasystole, the heterogenetic impulse as I have termed it, is intimately concerned in the disorders of greater magnitude. The hypothesis, carried so far, will, I think, meet with

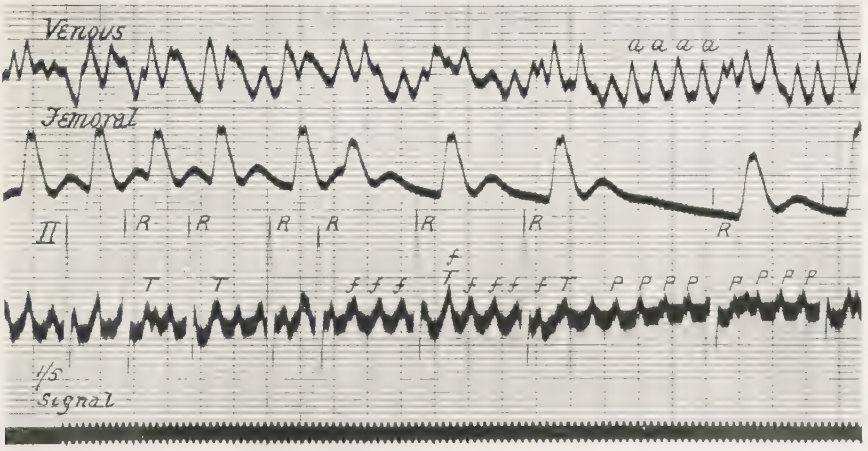


Fig. 307. Simultaneous venous, femoral and electrocardiographic curves from a dog, showing the passage of fibrillation to a disorder, which bears a close resemblance to clinical flutter, under vagal stimulation. Signal of vagal stimulation. Time in fifths of a second.

general assent; but it may not be left at that stage, for we have still to explain essential and obvious differences between one type of disorder and another, and in so doing we are upon far less solid ground.

CHAPTER XXVIII.

OBSERVATIONS UPON THE NATURE OF PAROXYSMAL TACHYCARDIA, FLUTTER AND FIBRILLATION.

The nature of paroxysmal tachycardia.—It is assumed that the paroxysms are in their essence serial extrasystoles. This explanation was originally put forward by Hoffmann (*321*) and subsequently abandoned (*322*) by him in favour of a hypothesis which is no longer tenable.* We come to the cause of repetition. Some years ago Wenckebach (*758*) attempted to isolate certain forms of cardiac "bigeminy" under the term "true bigeminy." He clearly recognised that the picture of paired beats arises in many instances as a result of premature contractions, each falling subsequent to a ventricular systole having a normal origin. He regarded such examples of paired beats as lying outside a rational definition of bigeminy, and desired to restrict the term to instances of twin beats which present certain definite characteristics. The qualities of an isolated couple of beats, which he deemed essential in order that it might be brought within the limits of the new definition, were two in number. First, that the second beat of the pair should lack a complete compensatory pause, and secondly, that the two beats should bear a constant time relation to each other. In a later publication (*760*) the definition was extended by the exclusion of the first qualifying factor. In brief, the definition was to include all instances of accurate coupling. In dealing with the subject Wenckebach distinctly implied that the term bigeminy, to be logically employed, must be restricted to such of the twin beats as may be supposed to be constituted by individual beats of an identical nature. This implies that where beats are accurately coupled the pairs consist of individual beats of an identical nature.

As I understand Wenckebach, he explained the accurate coupling by supposing that the two beats of the pairs are generated by the same impulse which is unduly prolonged; this view is inacceptable seeing that the two beats usually come from different foci in the heart, as galvanometric

* He sought to prove that the tachycardia represents the real heart rate in patients exhibiting paroxysms and that the periods of slow action are to be explained by heart-block. He stated that there is a simple mathematical ratio between the fast and slow rates. His curves failed to substantiate his statement, and his explanation is obviously inapplicable to paroxysms of simple tachycardia. Hoffmann's halving of ventricular rate is seen in cases of flutter, but these were not the cases which he attempted to explain.

examination of the patient almost always shows (*435*, *436*).* To explain accurate coupling† we are left with the obvious alternative that the second beat of the couple is in some way forced by the first. The moment we admit this hypothesis, and new observations have suggested it to the minds of several independent workers, the possibility that the second beat may force a third enters our conceptions. There are instances in which it is difficult to refuse an explanation of this kind.

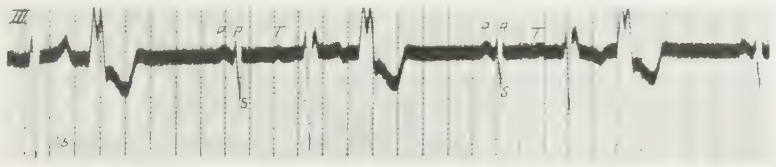


Fig. 308. ($\times \frac{2}{3}$.) An electrocardiogram showing a curious and continuous trigeminy of the heart of constant type. The normal cycle is followed at a fixed distance by an extrasystole starting in the left ventricle; this is then followed at a fixed distance by an extrasystole starting in the right ventricle. Time in fifths of a second.

Fig. 308 was taken from a patient who displayed a trigeminal beating of the heart. This curious mechanism was constant for considerable periods. Each normal cycle (*P*, *R*, *S*, *T*) is followed at a constant interval by an extrasystole of ventricular origin (left-sided); the extrasystole is in turn followed by a second of different origin (right-sided), and the group is then repeated. The order of the three beats is always the same, the intervals between them are repeated. The two extrasystoles in this instance could not be regarded as resulting from a single prolonged stimulus, unless we were also prepared to assume that this stimulus acted alternately at separate heart foci. Seeing that the second variety of extrasystole never occurred in this patient, except as a sequel to an extrasystole of the first variety, we must suppose that in some manner the first beat was responsible for it. If we are prepared to admit this causal relation as possible between beats arising from separate foci, we must be prepared to admit it as possible between extrasystoles which arise in the same focus, and this is the commoner event. The *hypothesis* as it stands is that in certain circumstances, as yet

* Wenckebach emphasised especially the bigeminy of the ventricle which is sometimes seen in complete dissociation. As I have shown (*435*), this coupling is extrasystolic in origin (see also Josúe and Godlewski (*358*).

† An explanation of triple rhythm, an extrasystole following two normal cycles, has been put forward by Flemming (*177*). He assumes two independent centres of impulse formation and that the impulses of the extrasystoles are built up at a third of the rate of the natural impulses. There are difficulties in accepting this hypothesis even when applied to triple rhythm; but the main argument against it is that it is inapplicable to many other forms of group beating and especially to short paroxysms of tachycardia. We are still quite in the dark as to why extrasystoles disturb a regular rhythm at one moment and fail to do so at another moment, seeing that the conditions at the two moments, in so far as they are to be observed, appear to be the same.

indeterminate, a single extrasystole may promote a second, and the second a third,* and that the process being repeated a paroxysm of tachycardia is engendered.

The nature of flutter.—Our knowledge of clinical auricular flutter is too incomplete to permit of more than speculation as to its exact nature. Reasons for believing that heterogenetic impulses stand to it in the relation of a causal factor have been adduced. It has also to be born steadily in mind that the condition is closely connected with fibrillation. It possesses a curious quality, namely, a tendency to persist in circumstances where the exciting cause appears to be no longer at work. In this it resembles fibrillation. Can we explain flutter on the simple basis of its being a paroxysm of extrasystoles of exceptional rate? The tendency for the paroxysm to persist could be explained on this basis, for there appears to be a critical rate which, if reached by a heart submitted to successive stimuli, tends to endure (*424*, *556*); further, an increase of rate will of course largely account in the case of auricular flutter for the disturbances of *A-V* conduction which are manifested in this condition. But, accepting this view, we should expect that the origin of flutter would vary, that the electrocardiogram would in its auricular elements present as variable a picture from case to case as it does in simple paroxysms. Now this is not the case; apart from exceptional instances, the auricular curves in flutter are wonderfully constant in form (see Fig. 256, page 267), and this uniformity has to be explained. If, therefore, flutter is in reality of the same nature as the simple paroxysm, it must be held to arise in some particularly sensitive region of the heart (such as the *S-A* node; see footnote to page 269) which determines both the relative constancy in the form of the auricular curve and the exceptional rate of the heart's action. An alternative suggestion brings me to describe an interesting phenomenon and view. It has been shown by Mines (*556*) that if a ring of tissue is cut from the auricle of a large ray fish, and if this ring is appropriately stimulated at one point, a contraction wave may be induced to propagate itself from that point in one direction only. When such a contraction wave is obtained it comes back to the point from which it starts and, the contraction (with its accompanying refractory state) having by this time subsided at the point stimulated, the travelling wave passes on and is continued as a movement which completes the circuit of the ring over and over again. Thus a series of contractions of the whole ring results from a single stimulus. This and similar ingenious demonstrations were employed by Mines to explain the persistence of *fibrillation* of the ventricle, in which he believed *circus* movement to play a large part, and his view has gained the adherence of Garrey (*211*) and Levy (*424*).† For reasons which I shall explain later, I do not consider this view, in the form in which it has been

* The further hypothesis of circus movement, which is clearly applicable to such repetition, is fully considered in the next section.

† See also the recent paper by McWilliam (*527*).

put forward, to be acceptable on the basis of our present knowledge; *if there is one disorder of the heart to which Mines' circus experiment would seem especially applicable, that disorder is flutter,** in which the heart cycles are complete and regular. Applied to flutter, circus movement would explain its persistence and it would also explain the contiguity of the auricular complexes (see page 268), seeing that an excitation wave would be travelling perpetually in the heart. The almost constant form of the auricular complexes from case to case would be explained independently of the focus from which impulses arise, for it would be a matter of indifference at what segment of the circuit they entered. Circus movement in the living heart would be determined, as those who have described it explain, by a reduction of the refractory period and delayed conduction (each of which would accompany very rapid heart action); it being supposed that circus movement arises when the refractory period becomes somewhat shorter than the total duration of spread of the excitation process. In other words it is suggested that, the auricle being stimulated to a greatly enhanced rate, there is a consequent reduction of the refractory period, and that the excitation wave is still spreading (and relatively slowly) in the muscle at a time when the first portion of the muscle to be thrown into the excited state is returning to rest and becoming non-refractory; what is not described by those who postulate the view of circus movement in the intact heart, is the path by which the re-entry is affected; despite its attractiveness, in this essential particular the conception is intangible. Before it emerges from the realm of hypothesis, it needs must become more clearly defined, and in the case of the ventricle take note in particular of the usual channels through which the excitation wave spreads.

The nature of fibrillation.—If it can be shown—it has not yet been shown though some suggestive evidence can be adduced—that circus movement is possible in the intact heart and underlies the regular and rapid movement of flutter, then unquestionably there will be a case for assuming

* There is an opening for misunderstanding here, for which inexactitude of terminology is responsible. Our concepts of pure flutter and pure fibrillation in the auricle are fairly clearly defined. Flutter in the ventricle, if it occurs, has not been named nor in detail described; fibrillation of the chamber is a term commonly applied to the disturbance over the whole period following cessation of the ventricular output or following the instant when, if stimulation is withdrawn, the disorder persists. Yet admittedly the records which are obtained from a faradised ventricle, shortly after it ceases to put forth blood or shortly after persistence of the disorder is established, are very different from those which are obtained from it in the later stages (see Chapter XXVI), and it is impossible to study these records without concluding that the mechanism of disorder is profoundly, though perhaps gradually, modified during the later periods. It seems to me eminently desirable that if confusion is to be avoided, the term fibrillation should be confined to the fully developed disorder, and that the earlier phases of the disorder should remain unnamed until they are more fully understood. It is possible that Mines had in view these earlier stages, in which the electrocardiogram displays relatively regular oscillations, but this does not appear in his exposition.

that circus movement may play a responsible part in provoking the more complex mechanism of fibrillation. For the two conditions are decidedly related.* But even if that assumption is conceded, there are still many which remain unexplained, for at the least it can be shown, I think, that fibrillation is not *purely* a circus movement. My view is that if "flutter" proves to be a circus movement, then a circus movement is readily converted into fibrillation. More than this I am not prepared to concede at present.

Incidentally there seems to be no satisfactory reason for believing that fibrillation in auricle and ventricle are essentially different, or that such differences as appear to exist between them are not ascribable to differences in their sizes, their muscular architecture and in the system of conduction in the two chambers.

In attempting to conceive the manner in which fibrillation is provoked, I favour the hypothesis introduced by Engelmann (*129*) in the case of the ventricle; it is that impulses are built up independently in a number of separate foci in the ventricle. There is much to support this view.

I have already remarked that simpler forms of irregularity, unquestionably of heterogenetic (extrasystolic) origin, pave the way to fibrillation, however induced. Certain of these preliminary irregularities are remarkable. Before the ventricle fibrillates in response to the poison chloroform it is very customary to see a simple ventricular paroxysm develop. This paroxysm may at first consist of beats propagated from a single focus, but sooner or later this gives place to a more complex disturbance. Beats are propagated from other foci and intermingle (see Fig. 297, page 316). Not infrequently, as a direct precursor of those events which lead up to the earliest phases of persistent disorder, this shuffling of different types of contraction becomes extreme; yet the mechanism is still capable of analysis and is recognised as one in which very many regions of the ventricular muscle are contending together to master the rhythm of the heart. In instances in which the onset of clinical auricular fibrillation has been recorded a similar train of events has usually been distinguishable (*487*, *698*). In my own patient a short and regular paroxym of rapid beats started in an auricular focus; after a time this paroxysm was itself disturbed by premature beats arising in a separate auricular focus; this new disturbance became more frequent and insistent immediately before the auricle fibrillated. The events which culminate in fibrillation and with which we are most conversant, are first, the propagation of contractions of extrasystolic type, and secondly, their propagation from several or many points.

Any hypothesis put forward to explain fibrillation of the auricle, must account for a cardinal feature of the condition, namely, the complete

* It has been supposed that the coarsely irregular movements which the auricle may show at times is an admixture of flutter and fibrillation (Robinson, *647*, *648*). This view would confine the term fibrillation in the auricles to the finely divided movement, a definition which has a good deal to recommend it.

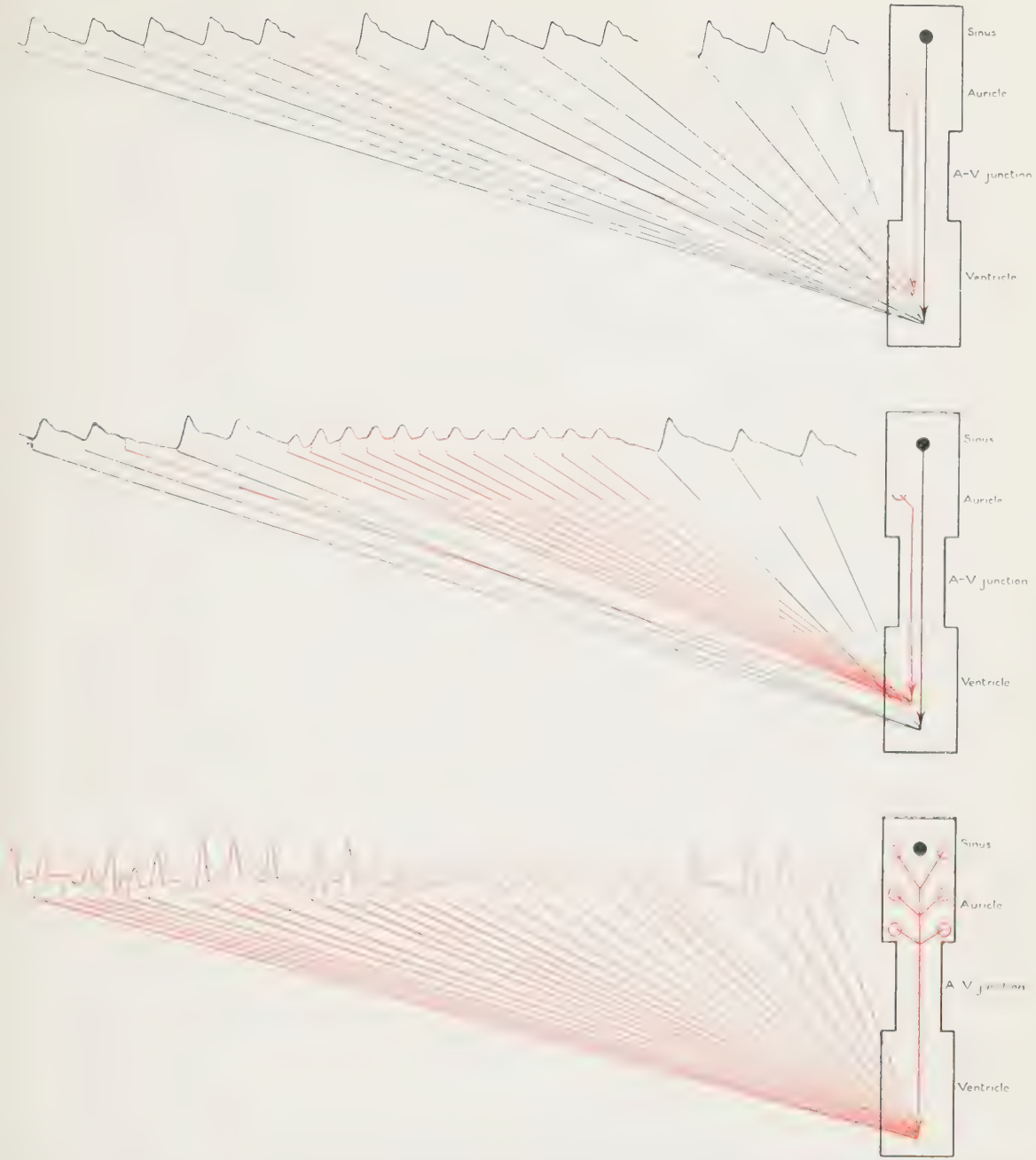


Fig. 309. A diagram illustrating the view of the close pathogenetic relation between the single extrasystole, paroxysmal tachycardia and fibrillation, as they occur in the auricle. The heart is diagrammatised to the right, and the *black* and *red* arrows and their continuations indicate the sources of the beats which are *homogenetic* and *heterogenetic*, respectively. In the uppermost pulse curve two premature beats are shown. In the middle curve a single premature beat and a short paroxysm of tachycardia are observed. In the lowest pulse curve the complete irregularity associated with auricular fibrillation is depicted. The red beats are considered to be of heterogenetic origin. Actual pulse curves have been utilised in constructing the figure. The view adopted is that the three mechanisms are provoked by one and the same process, and that the higher grades of disorder are brought about, either by an increase in the exciting agent or by an increase in the irritability of the tissue responding to it. NOTE.—It is to be observed that I show the heterogenetic beats as derived from *ectopic* foci; they are usually so derived, but not necessarily so

irregularity of the ventricle. Now this response of the ventricle is not unique, for, as Garrey (*208*) has pointed out, when the ventricle fibrillates the auricles beat irregularly. The impulses from the fibrillating chamber, be it auricle or ventricle, are conveyed through the auriculo-ventricular bundle (*76, 187*). It is not the peculiar constitution of this bundle or the admixture in it of muscle and nerve fibres which is responsible, for a similar phenomenon may be observed in the auricle itself. If the appendix of the auricle is made to fibrillate and its connection with the remainder of the auricular tissue is broken except over a narrow bridge, the remainder of the auricle beats irregularly in response to the fibrillating appendix (*459*). Whether the response of a muscle mass to a neighbouring and fibrillating mass, which is connected with it, is by fibrillation or by a series of irregular contractions, depends purely on the cross section of the connecting bridge. If there is sufficient tissue fibrillation will be conveyed as such, if there is less tissue in the bridge irregular impulses will be conveyed. I explain the phenomena by supposing that in the fibrillating appendix impulses are built up rapidly and in separate regions; it is the clash of the separate contraction waves in their pursuit of different courses in the appendix, which produces the turmoil and destroys co-ordinate contraction. From this turmoil the bridge receives a number of distinct impulses in the shape of excitation waves; the number received will be relatively few where the bridge is narrow, moreover the impulses will tend to impinge upon it as waves set parallel to the cross section of the bridge and will consequently follow each other across the bridge without mutual interference. But should the bridge be wider, then the waves will be correspondingly more numerous, they will enter at different angles, will meet in the bridge and produce in it a fibrillation similar to that from which they are derived. The observations which Garrey* uses to support the hypothesis of circus movement, namely, that fibrillation only occurs in muscle of sufficient mass, and is more complex the greater the mass (*211*), seem to me to harmonise equally with the view which I support; for the larger the mass, the more foci are there to liberate impulses and the more paths are there for the waves to pursue.

If fibrillation is dependent upon circus movement, it may be asked how the circus movement is conveyed from fibrillating appendix to auricle through a bridge of half a centimetre's width?† Does the appendix set up a circus movement in the bridge and does this in turn promote a third circus movement in the auricle proper?

This experiment is not explicable purely on the basis of simple circus movement, neither, so far as I can see, is the irregular but co-ordinate response of muscle guarded by a narrow bridge.

* Garrey also supposes that heart-block takes a large share in the disorder.

† Garrey concludes that the fibrillating movements in a distant portion of tissue are not sustained from the point at which fibrillation is initiated by stimulation.

There is finally an experiment (*468*) of a different kind, the results of which are quite compatible with the hypothesis of multiple foci and independently contracting areas, but, so it seems to me, inconsistent with that of simple circus movement.* Two small contacts are laid side by side on the wall of the ventricle, each is paired with an indifferent point and from each pair of contacts a galvanometric curve is taken. The two areas of muscle so investigated are adjacent, their centres lie but a few millimetres apart. The curves are taken simultaneously and, while the heart is beating normally, are almost indistinguishable in form and almost simultaneous in their time phases. The two areas are activated co-ordinately. If the ventricle is now stimulated with a faradic current some distance away, the heart when it first responds does so by a series of rapid contractions at a rate of some 300 or less per minute. The ventricle becomes embarrassed but it is not yet fibrillating for, if the electrodes are lifted, the beats at once discontinue; the records during this rapid heart action remain alike and the two areas show almost simultaneous activity; the activity, as between them, is still co-ordinate. But press the stimulation further and the "first stage of fibrillation" supervenes. The deflections become irregular in amplitude and incidence. Their rate is now 500-600 per minute, and although each record yields a series of this kind, yet in the two records there is no longer correspondence of amplitude, neither is there any longer accurate correspondence of the times of the deflections. As the fibrillation proceeds to its full development the oscillations become slower and more irregular and the lack of correspondence between the curves becomes more pronounced. There may be an incomplete sympathy in the early stage, there is less in the final stage. Co-ordination between fibres so little removed from each other as they are in this experiment appears to be at least in large measure lost when the mass of muscle passes into fibrillation.†

It seems certain therefore that fibrillation is not a simple circus movement, and that, if a circus movement (or movements) is actually present in the fibrillating muscle, other factors also play a large part. It should be remarked that, whether circus movement is introduced into the hypothesis or not, fibrillation in its stage of initiation is to be regarded as due to the elaboration of new impulses. For it is necessary to assume the birth of such impulses to start a circus movement. The problem would be much simplified if, by common consent, the possible relation of circus movement only to flutter came under discussion, and if the relation between flutter and fibrillation were to be held for the moment as a distinct question, until the first question might be regarded as settled.

* And equally inconsistent with Rothberger and Winterberg's hypothesis (*674*) that fibrillation is conditioned by a reduction of the refractory period and consists simply of an extreme acceleration of rate.

† The degree in which it is lost is not yet clear; further experiments seem desirable and are actually in progress.

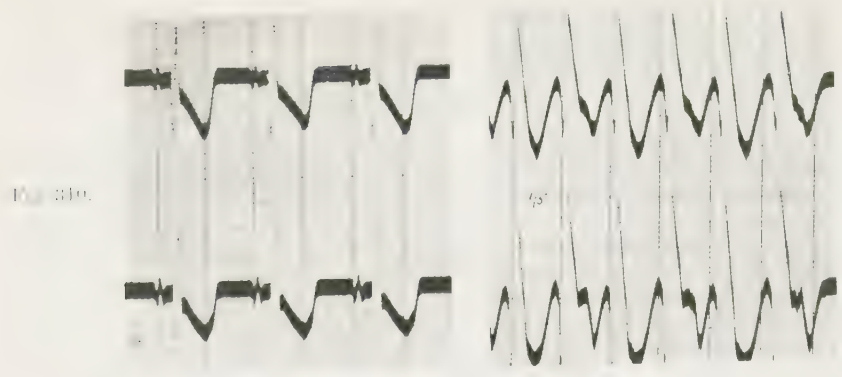


Fig. 310. Fig. 311

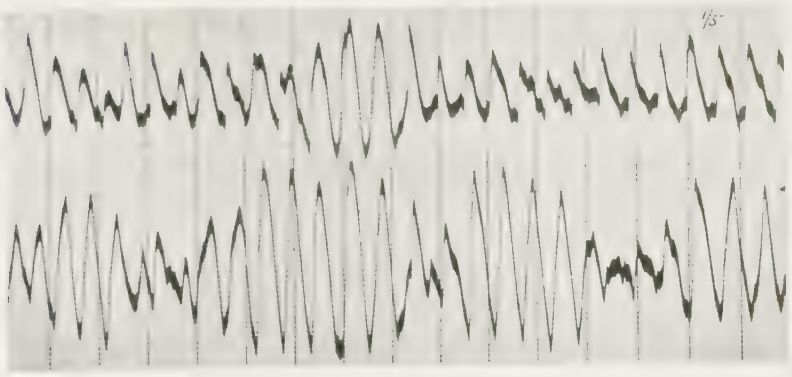


Fig. 312.

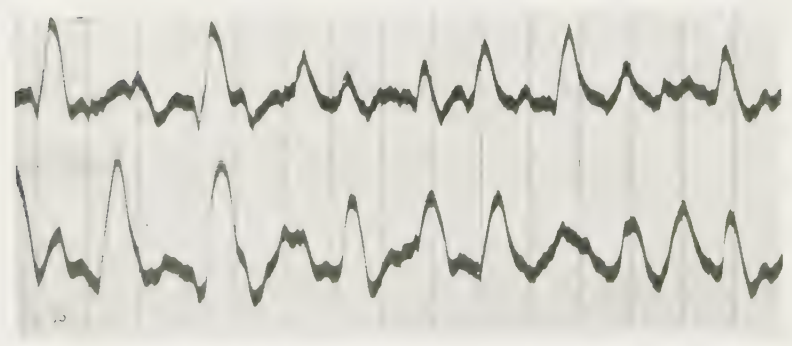


Fig. 313.

Fig. 310-313. (× ⅔.) A series of simultaneous electrograms taken from closely adjacent contacts on the surface of a dog's ventricle. In Fig. 310 the heart is beating normally and the two curves correspond, resembling each other in all phases and being practically simultaneous in all phases. At this stage the adjoining areas of tested muscle are activated coordinately. In Fig. 311 the heart is responding to a faradic current by means of a series of regular beats at a rate of 290 per minute; persistence of the disorder at the withdrawal of stimulation is not yet established and the curves are still in correspondence. Note the presence of concordant alternation in the two curves. In Fig. 312 persistent disorder has become recently established. The curves are no longer regular, neither do they harmonise with each other in respect of the amplitude of the oscillations or the precise time relations of the oscillations. In Fig. 313 the final phase fibrillation, properly so termed, is present. The disorder between the oscillations is now more striking. Time in fifths of a second.

It may still be held, I think, that the formation of stimuli at multiple foci and the starting of independent contractions will account for all, or at the least almost all, the phenomena of fibrillation in auricles or ventricles. That there may be coincidentally a reduction of the refractory period and also a lessened power to conduct is probably, almost certainly, the case. To those who regard increased "irritability" as the fundamental cause of fibrillation, meaning by "irritability" the power to produce new impulses, the reduction of the refractory period and lessened conduction* are regarded as secondary and consequent events. However, the view cannot be held without reserve. It is said that many of those influences which tend to increase the refractory period or to decrease rate of conduction tend to abolish fibrillation as such (*527, 674*). Direct observation of the fibrillating ventricle strongly suggests the appearance of numerous and changing lines at which contraction waves cease abruptly; influences which are known to depress conduction (for example, vagal stimulation in the case of the auricle) tend usually to perpetuate fibrillation or to increase its intensity. Gaskell regarded block as an important factor in promoting fibrillation. The precise part played by these factors, namely, the refractory period and conduction must for the moment be left *sub judice*. The real nature of fibrillation is still unknown; our explanations remain in the stage of hypothesis.

* In respect of conduction, it is to be pointed out that we have little or no knowledge of influences affecting conduction in the auricular or ventricular muscle; our knowledge is almost confined so far as the mammalian heart is concerned to changes of conduction in the *A-V* system, an important distinction which is often overlooked.

CHAPTER XXIX.

CAUSES DETERMINING EXTRASYSTOLES AND ALLIED DISORDERS.

MECHANICAL records of the heart, whose beat is disturbed on the one hand by spontaneous extrasystoles and on the other hand by premature responses of the heart to external stimulation, are alike in every respect. The electrical records differ chiefly in that those corresponding to external stimulation are of greater variety, for the points from which they may be obtained are innumerable. We have but incomplete knowledge of the tissues from which extrasystoles originate. Electrical records seem to show them arising in the *S-A* and *A-V* node; the system of Purkinje fibres is suspected to be responsible for some which come from the ventricle. But the shapes of many of the electrical complexes associated with auricular extrasystoles suggest the mass of the auricular muscle as an occasional if not a frequent point of origin. Briefly, extrasystoles cannot be associated exclusively with one or other class of tissue.

That the extrasystole and the forced beat have much in common is obvious; the processes which underlie the extrasystoles are of short duration, the response of the heart to an induction shock is almost immediate. Possibly we are on safe ground in assuming that the external stimulus takes the place of the process which awakens an extrasystole; if that is so then the after events are in the two cases identical. But we know too little of the process in question to speculate as to its nature, further than to suppose that a stimulus of some special kind is born in the muscle. When Wenckebach (*758*) classes extrasystoles under disturbances of excitability, the analogy with artificially forced beats is driven too far. Excitability is the attribute of muscle which governs its response to electrical and mechanical stimulation.* The ventricle is said to be hyperexcitable when it responds to a stimulus which for the normal heart is below threshold value. A rise of excitability cannot be conceived to originate an extrasystole, unless it is assumed further that continuous or discontinuous impulses, below threshold value, are a constant phenomenon in normal heart muscle. Almost certainly this is not the case; it is much more probable that extrasystoles are conditioned by

* Further, as Hering (*289*) has quite properly pointed out, we may not assume that the heart's sensitivity to electrical excitation is necessarily a measure of its sensitivity to impulses formed intrinsically.

effective stimuli newly generated in the muscle; for excitability as tested electrically may be found diminished in hearts which at the time exhibit spontaneous extrasystoles: this is so when the heart is poisoned by potassium chloride: and the excitability of the ventricle may be greatly raised without the appearance of extrasystoles.

In reviewing the ways in which premature beats are induced experimentally, the distinction between these responses and the spontaneous extrasystole must not be forgotten. Premature beats may be induced by many forms of external interference, by submitting the muscle to electrical stimuli, such as induced shocks, the makes or breaks of a constant current, or the passage of the constant current itself if this is of sufficient strength. They follow mechanical stimulation, such as sharp taps or pin pricks, they follow when a chemical irritant, such as a crystal of salt or a drop of acid, is placed on the muscle. They may be induced also by a sudden and local rise of temperature. Most of these methods, suitably controlled, will bring forth the solitary extrasystole, a group of forced beats or in the end fibrillation; though the reaction of the heart varies a good deal to the same stimulus in different circumstances. This variation we describe as a variation of excitability; such is the definition of our term.

But extrasystoles, short paroxysms of rapid heart action and fibrillation may be provoked by apparently quite different causes. Thus, it has been recorded that either of the two first-named disturbances will sometimes follow an abrupt rise of ventricular pressure, such as follows clamping of the aorta (*252*). There may be an association with anæmia, as when extrasystoles follow the sudden obstruction of the venous inlets (*705*),* or when any or all of these three disorders follow occlusion of a coronary artery (*432*). All may be provoked by the intravenous injection of poisons, notable amongst which are chloroform (*426*),† digitalis (*85*, *506*, *507*), strophanthin (*672*), adrenalin (*360*), nicotine (*594*), potassium salts (*546*), barium chloride (*668*), and aconitine (*87*). Extrasystoles have also been seen in muscarine and physostigmine poisoning (*663*). In these cardiac reactions variability is also the rule, but here we may not speak of variable excitability, for the impulses which force the new contractions are not apparent. These disorders are carefully distinguished by referring them to increased *irritability*, the term being used in such a sense that it implies no more than the observed facts.‡

In the human subject the origin of extrasystoles and allied disorders is often more obscure still. The simple disturbance (extrasystole) is

* A clinical parallel to this experiment is found in cases where paroxysms of tachycardia or extrasystoles are seen only in the erect posture, the blood tending to accumulate in the lower parts of the body.

† Rothberger and Winterberg (*576*), have been unable to obtain these irregularities by administering small doses of chloroform, (see however Levy, *Heart, 1919, VII, 105*).

‡ If we ascribe them to heightened excitability, then amongst other things we must assume gratuitously a lowering of the threshold response to stimulation,

extremely common, so common indeed that the more its prevalence is investigated the more certain does it seem that all men are subject to it from time to time. Unquestionably its incidence is most frequent in patients in whom there is structural disease of the heart, but its frequent occurrence amongst apparently healthy people largely destroys the significance of this relation. Paroxysmal tachycardia is much less frequent, and while it is also found in people who otherwise appear healthy, it is much more prevalent in heart disease. Auricular fibrillation and flutter are regarded, I think universally, as invariable signs of heart disease. Having noted the prevalence of these types in their general relation to heart disease, we may particularise. An attempt to associate extrasystoles with any special lesion of the heart muscle has for obvious reasons proved unfruitful. Lesions in the muscle, not dissimilar to those found in fibrillation, have been described in a few cases of paroxysmal tachycardia. The hearts of those dying with auricular fibrillation have been investigated much more thoroughly. In the earlier days, when complete irregularity of the heart was thought to be possibly of the nature of sino-auricular block (*761*), morbid anatomists sought and found lesions in a bundle of muscle fibres which Wenckebach had described as coursing from superior vena cava across the sulcus (*247*, *692*, *693*). At a later date the *S-A* node was discovered, and in turn this structure was examined and profound lesions were often found in it (*386*). But wider search revealed lesions of the same class in other parts of the auricle, notably in the *A-V* node ; moreover in many of these hearts equally distinct lesions were seen scattered in the ventricle. At present it is recognised that the type of lesion associated with auricular fibrillation is inconstant (*497*), but that scattered foci of chronic inflammation are the rule, and that these, while rarely confined to particular structures, congregate in the auricle, especially in those parts of the auricle which contain special tissue. Similar lesions are discovered in the hearts of patients not affected by fibrillation (*567*). In other hearts affected by the disorder the lesions are not seen (*497*) and this is notably so in the case of the horse (*58*).

To sum up, the appearance of the tissues throws no real light upon the cause of the cardiac disturbance. Fibrillation of the auricle, like the simple paroxysm, may be transient ; it is scarcely to be anticipated that tissues taken from these hearts during attacks or during periods of quiescence would be distinguishable.

The only single disturbance of the group which can reasonably be associated with a structural change is fibrillation of the ventricle in cases of sudden death following embolism or other sudden occlusion of a coronary artery.

Although extrasystoles are frequently associated with high blood pressure, this fact is not to be correlated with their appearance in experiment on clamping the aorta. To particularise in this manner is to neglect the broad questions at issue. It is a common belief in clinical circles that many

irregularities of the heart are due to over-distension of its chambers and especially that fibrillation of the auricles may be so provoked. This conception, a legacy from tradition, is purely speculative. Fibrillation of the auricles is often seen where there is no dilatation, it is often absent where dilatation exists; moreover, experimental dilatation of the heart does not produce it. The only known relation between fibrillation and dilatation is of the converse kind; when the heart muscle is diseased or exhausted, the onset of fibrillation may lead to dilatation by embarrassing the heart in its work.

The rôle of the heart nerves in causing extrasystolic irregularities.

Irregularity of the heart has long been regarded by many as resulting primarily from derangement of the cardiac nerves. This general view, lacking sufficient evidence, was fanciful when it was put forth. Of recent years the rôle of the heart nerves has been the subject of much careful work; although knowledge remains incomplete and some of the evidence is still conflicting, we are beginning to understand the relation of the heart nerves to the individual forms of irregularity as they are now recognised. At present we deal with the largest group, namely, those which are frankly extrasystolic and those which are allied. First of all, it is to be emphasised that each of these irregularities may be seen in the isolated and perfused heart, all connections between heart and central nervous system being severed. Thus, it is known that extrasystole, paroxysms and fibrillation may be determined by changes in a heart freed from all central nervous control. Numerous attempts to produce such irregularities of the heart by stimulating, or by otherwise interfering with, the vagus and sympathetic nerves, have been uniformly unsuccessful until recently. Interest in the nerves was reawakened by Rothberger and Winterberg who claimed to have induced extrasystoles by combined stimulation of the vagus and sympathetic nerves (*667*). Close scrutiny of their published curves does not support this claim (*468*); the beats being escaped contractions and not extrasystoles*.

Out of a large number of experiments, in which the sympathetic was stimulated while vagal tone was high, Kuré (*403*) figures two instances in which an extrasystole appeared. Excepting such examples, I can recall no evidence that simple or combined stimulation of the cardiac nerves can by itself awaken either single or successive systoles. But Rothberger and Winterberg also state that fibrillation of the ventricle may be produced in a similar way. Their interpretations of their published curves is unexceptionable; yet fibrillation of auricle or ventricle is not a very

* The curves purport to show "extrasystolic" tachycardias, actually they show beats in rhythmic succession at a relatively low rate, and emanating probably from A-V node or ventricle.

uncommon accident in animals under experimental conditions, and this paper is not the first to report fibrillation as a sequel to interference with the vagus. The appearance of this disorder is most capricious. Now if, as in their series of thirty experiments, fibrillation was the response on but three occasions, then clearly vagal stimulation, so often repeated without success, can be regarded as no more than one, and probably by no means the chief causative factor. The same criticism applies to Kuré's example.* If we are to conclude that nervous impulses, acting alone, are capable of producing extrasystoles or an allied disorder of the heart beat, stronger evidence than occasional reactions is needed. The usual failure of the reaction calls for explanation. I have myself (*466*) seen groups of extrasystoles repeatedly follow vagal stimulation in a single experiment (Fig. 314), but this experiment

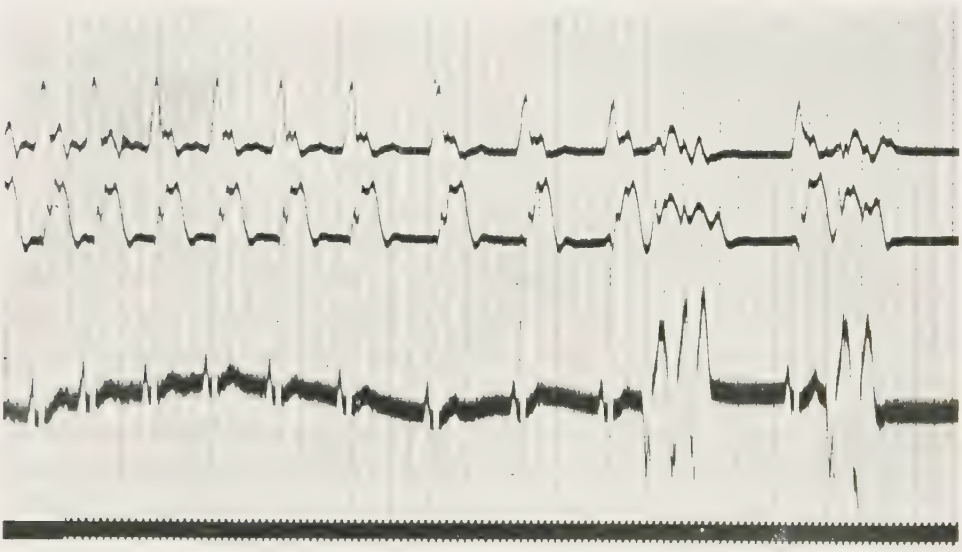


Fig. 314. (*Heart, 1913-14, V, 247, Fig. 13.*) Simultaneous myocardiograms of auricle (top curve) and ventricle (middle curve) and electrocardiograph, from a dog (lead *II*), showing an exceptional response of the heart to stimulation of the left vagus. The vagus was repeatedly stimulated with the same result in this animal, but the reaction was only obtained at one phase of the experiment. The signal of stimulation is shown below. Time in fifths of a second.

also stands by itself, although I have stimulated the vagi under many different experimental conditions on hundreds of occasions. It is not permissible to conclude from isolated experiments that an impulse travelling along a cardiac nerve is by itself responsible for an extrasystole ; it is more

* See also Hering (*290*) and Rihl (*639*).

legitimate to conclude that into such experiments some unrecognised factor has obtruded itself. If an experiment could be devised in which simple interference with the nerves was invariably followed by extrasystoles, the case would wear a different complexion. The causes of extrasystole are known to be elusive ; they creep into experiments unseen and unheeded. Isolated examples such as have been cited are attributable in the first place to the unseen factor, and only in the second place to altered innervation which tips the balance.

These remarks bring us to Rothberger and Winterberg's later work (*668*) and to that of Levy (*422-426*). The former workers have shown that stimulation of the sympathetic nerves is followed by beats of an extrasystolic nature, *when previously the heart has been poisoned* by small doses of barium chloride. Levy has shown that stimulation of the sympathetic (direct or reflex) or section of the vagi* has the same effect when the heart is poisoned by small doses of chloroform. The evidence for both these statements is conclusive. In each of these series of experiments a drug (barium chloride or chloroform) is employed which is known to induce an irritable condition of the heart, predisposing to or actually eliciting extrasystoles. In certain circumstances, as we have seen, a sudden rise of blood pressure is followed in experiment by extrasystoles. According to Weiland (*750*), vagal stimulation facilitates their production by this means. Thus it is to be acknowledged that the nerves of the heart may play a rôle in provoking the disorders of mechanism considered. Can we define this rôle more distinctly ? There are, so it seems, two questions to which answers may be returned at the present time. Has it been shown that abnormal nerve impulses playing upon a normally nourished heart may be responsible for extrasystoles or the higher types of disorders ? To this question we may return a negative answer. May nervous impulses playing upon an irritable organ provoke these curious contractions ? To this question we may return an answer in the affirmative.†

A clinical example of a parallel kind to those cited has been described in a patient in whom extrasystoles and paroxysms of tachycardia were prevalent. In this case Kuré (*402*) was able to induce attacks with regularity by setting the girl sums in mental arithmetic. I have seen a similar though less striking example. That attacks of paroxysmal tachycardia may be provoked by emotional excitement in those who are predisposed to them is well known.

* The abolition of vagal tone may provoke paroxysms of tachycardia on occasion, as in Galli's patient, where paroxysms *in a patient subject to them* frequently followed atropine injections (unpublished).

† For further discussion of this subject see also *288* and *290*.

CHAPTER XXX.

SINO-AURICULAR BLOCK, SO-CALLED.

THERE is a peculiar disturbance of the heart's action, first graphically recorded by Mackenzie (*502*, *Fig. 5*) in 1902, to which the term *sino-auricular block* has been applied provisionally. In its simplest recognisable form it consists of dropped beats, thus resembling an early stage of auriculo-ventricular block; but it differs from the latter in that the auricular beat is lost as well as the ventricular. When this apparent loss of a whole heart beat happens, the rhythmic action of both auricle and ventricle is disturbed by a cycle of unusual length, the long cycle being approximately double the length of rhythmic cycles. I say approximately advisedly, for the cycle in question is usually somewhat shorter than two normal cycles; moreover, it is ushered in by slight quickening of the whole heart and is succeeded by cycles which, while at first a little long, shorten up until the usual length of cycle is re-established.

It was this arrangement of the heart cycles which originally led Wenckebach (*760*) to suggest the current explanation of the irregularity. To explain the curves which he published he used auriculo-ventricular block as an analogy. It will be remembered that in auriculo-ventricular block, a "dropped beat" is rarely uncomplicated, being usually associated with preliminary and subsequent changes in the lengths of the *A-V* intervals. These changes in the intervals alter the disposition of the ventricular beats in so characteristic a manner (see Fig. 124, page 173) that the presence of block (as opposed to extrasystole) may be recognised in arterial curves; in point of fact heart-block was first identified in the human subject by purely arterial signs. In simple instances of the irregularity described in this chapter, the disposition of the ventricular beats is identical with that found in auriculo-ventricular block, but the recorded beats of the auricle are not undisturbedly rhythmic as they are in the last-named condition; the auricle participates in the irregularity equally with the ventricle. In auriculo-ventricular block the irregularity is manifested by the ventricle only and changes in conduction at the *A-V* junction fully explain it; to explain a similar irregularity of the auricle—the ventricular irregularity in this condition requires no further explanation than that the ventricle follows the auricle passively—heart-block is still invoked, but the deficiency in conduction is supposed to occur at a higher heart level, namely, the junction of sinus and auricle; whence comes the term "sino-auricular" block.

The irregularity and the hypothesis which attempts to explain it may be illustrated by the polygraphic curves and diagram of Fig. 315. The arterial pulse in this curve presents frequent intermissions ; the long cycles are less than double the length of the short cycle, as in *A-V* block ; the short cycles decrease in length as they succeed each other. But when the venous curve is examined, the anticipated *a* waves are not found in the long diastoles ; the auricle participates in the irregularity ; the ventricle simply repeats the contractions of the auricle.

To explain such a curve it is supposed that the impulses liberated by the pacemaker are rhythmic, but the conduction intervals to the auricle (*S-A* intervals) are conceived as varying in length according to the duration of the preceding periods of rest (see diagram below Fig. 315).

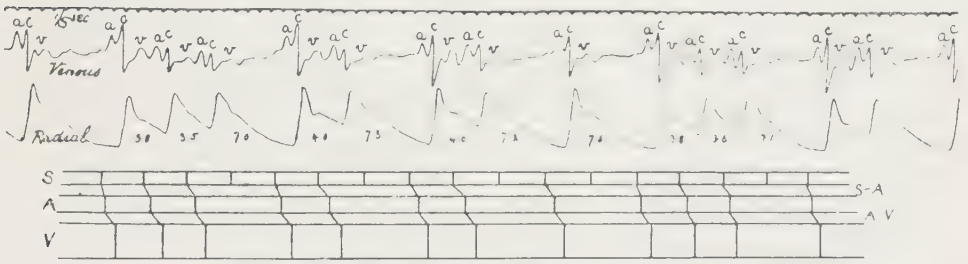


Fig. 315. (× $\frac{3}{4}$.) Polygraphic curve showing a sinus irregularity during a period of suspended respiration. The arterial curve resembles those found in partial *A-V* block ; the venous curve shows that the auricle participates in the irregularity. The diagram reads the events of the heart upon the hypothesis of " sino-auricular " block.

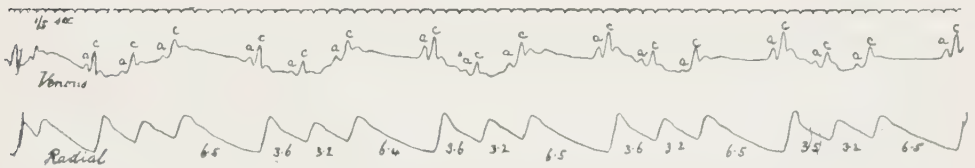


Fig. 316. (× $\frac{3}{4}$.) Polygraphic curve from the same patient, and showing the same type of irregularity. In this curve the irregularity is clearly associated with the acts of breathing, the whole venous curve dips and the pulse quickens with each inspiration. The pulse became regular for the first time when atropine was administered.

Many similar irregularities have now been published (*158*, *306*, *419*, *620*, *631*, *709*). The irregularity may result from digitalis administration (*306*) (Fig. 317). In not a few patients it appears to be associated definitely with inhibitory influences ; this association was notable in the case reported for me by Stokes (*709*), in which from time to time the short cycles fell solely in the inspiratory periods and the long cycles in the expiratory periods of respiration. A curve from this case is shown in Fig. 316, in which

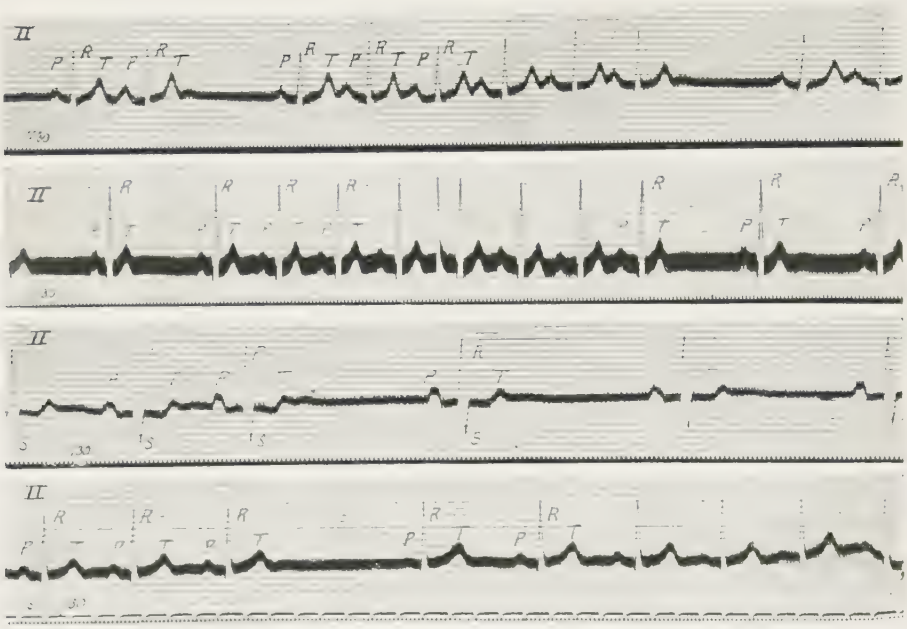


Fig. 317. ($\times \frac{4}{5}$.) Four electrocardiograms from separate patients, to illustrate the condition termed "sino-auricular block." The time is in thirtieths of a second.

In the first curve two intermissions of auricle and ventricle are seen. Note the increased length of most of the *P-R* intervals.

In the second curve a slow heart action gives place abruptly to an action almost twice as rapid; after eight cycles, the first of which shows auricular quickening, the auricular rate drops abruptly and to exactly half its former rate. The upstroke between the fourth and fifth rapid beats was due to an escape of extraneous current into the instrument. From a case of exophthalmic goitre. Note the varying *P-R* intervals.

In the third curve the heart rate drops abruptly to almost half its former rate. The auricular rate is increasing directly before the heart rate falls. This curve was taken from a case of mitral stenosis in which the *P-R* interval was long and in which the irregularity resulted from digitalis.

In the fourth and last curve a solitary intermission of auricular and ventricular systoles, succeeded, first by slowing, then by quickening of the auricular rhythm, is seen.

inspiration is to be recognised by the dips of the venous curve as a whole. In Danielopolu's patient (*99*), as in Stokes, the heart action became natural after the administration of atropine. The suggestion of vagal responsibility is also supported by the observation of similar heart irregularities in man as a result of deliberate vagal stimulation (*631*).

This irregularity, provisionally termed sino-auricular block, is responsible for several forms of disordered heart action. It may manifest itself in simultaneous intermissions of auricle and ventricle and these may be isolated (Fig. 317, first and last curve); it may be responsible for grouping of the pulse beats (Fig. 315 and 316); it may give rise to abrupt slowing of the

whole heart (Fig. 317, middle curves). Sometimes this slowing consists of an abrupt and exact halving of the whole heart rate ; in the second curve of Fig. 317, this halving is well demonstrated by the auricle. But more often the slow rate is slightly more than half the fast rate, each of the long cycles being less than two of the short cycles taken together, as is the case in the third curve of Fig. 317 ; this is attributable, as Levine has pointed out, to quickening of the sinus rhythm, quickening which is to be identified by measuring the last cycle of the fast rhythm (this shows shortening) and which is responsible for the change from fast to slow rate of auricular beating (*i.e.*, is responsible for the " block "). Finally, the same form of disturbance is probably responsible for occasional instances of profound and continued slow action of the whole heart. In the cases to which I refer the heart rate may be as low as 30 beats per minute. The electric curves (of which Fig. 318 is an illustration) show natural heart cycles separated by long diastoles. This slow action is distinguished from other forms of profound slowing of the whole heart by the fashion in which the heart accelerates in response to exercise. The rate does not rise gradually ; it doubles abruptly, subsequently

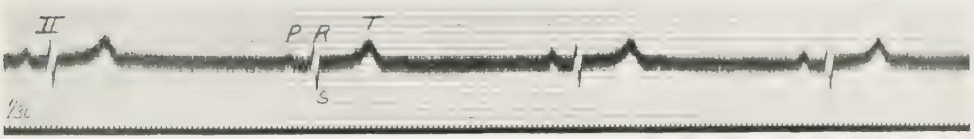


Fig. 318. An electrocardiogram taken from an athlete while at rest, and showing a regular action of the whole heart at 36 per minute. With moderate exercise the rate rose suddenly to double the original rate. The slowing was probably the result of " sino-auricular block." Time in thirtieths of a second.

accelerating gradually as does the normal heart in response to effort. After exercise the rate gradually falls and, when it has fallen to 80 or 70, it halves abruptly and the original slow rate is restored. Levine (*419*) has recently recorded a case in which he believes that several successive sinus impulses were blocked from time to time.

So-called sino-auricular block frequently consorts with auriculo-ventricular block. Of the four electrocardiographic curves now published (Fig. 317), three show distinct impairment of *A-V* conduction as evidenced by a prolonged *P-R* interval ; this interval shortens again immediately after each long diastole. In many of the recorded cases (*306*, *502*, *631*, *760*) one intermission may involve the auricle and ventricle while the next involves the ventricle only. Frequent intermissions of the two kinds may be mixed together and are then responsible for very complex irregularities. The association of two cardiac disorders, inter-related in other ways, is too frequent to be regarded as accidental.

General remarks.

If a ligature is tied around the sinus of the frog's heart (1st Stannius ligature) the heart ceases to beat below the level of the ligature, whereas above it the rhythmic movements of the veins are still to be seen. This experiment and others have led to the view that the beat originates in the veins and spreads from them to sinus proper and so to the auricle and ventricle. The exact location of the pacemaking tissue in the lower vertebrates remains uncertain; recent work indicates that the contraction starts in the sinus and spreads against the flow of blood up the veins (*549*). It is clear, however, that the 1st Stannius ligature is placed below the level at which the rhythm of the heart is generated. Evidently any lesion of the heart which damages the whole circumference of the tube in this region will lead to disturbed beating of the heart below the level of the damage. It is not difficult to devise experiments in which the mammalian pacemaker is isolated, or in which it is cut off on three sides from the rest of the auricular tissue while the remaining connecting bridge is impaired by injury; and in such experiments disturbed beating of the heart similar to the clinical disorder will result. Such experiments do not illuminate the clinical condition, for it is scarcely to be conceived that the latter is produced by lesions at all comparable to those effected artificially.* Continued beating of the veins, while the auricle and ventricle stand still, is frequent in the cold-blooded heart while dying or while it is under vagal inhibition; it occurs therefore in circumstances other than those of direct damage. The phenomenon, at first attributed to a natural line of block between the veins and auricle, is not understood fully.

When it was thought that the mammalian heart beat originates in the great veins, a connecting link between these and the body of the auricle was sought and apparently found in a fasciculus of muscle coursing from the superior cava; assuming this muscle band to convey the impulse, a clear conception of sino-auricular block was possible (*760*). But this conception became untenable when further observations upon this region of the heart had been undertaken. In the mammalian heart there is no true sinus, it is incorporated in the auricle; the contraction is now known to start in the sino-auricular node and to spread in all directions from this structure into the auricle and against the blood stream up the superior cava. I can find no sufficient evidence, anatomical or physiological, of a special and confined path of spread between node and auricular tissue; there is no distinct strand susceptible to damage by a local lesion. Thus, a cut or crush traversing the tail of the node is without perceptible effect, as I can assert; and it has been

* In Erlanger and Blackman's experiment (*147*) a ligature was passed from the inferior to the superior cava and twisted upon itself. Irregularities of the heart comparable to sino-auricular block followed. But the manner in which this effect was produced is not quite clear; presumably the ligature interfered directly with the pacemaker.

shown (*71*) that the node requires to be isolated on four sides before the auricular rhythm is affected. On the other hand Eyster and Meek believe that special and separate paths from the *S-A* node to auricle and ventricle exist and that "sino-auricular" and "sino-ventricular" block as they term the conditions occur when these paths perform their function improperly (*164*). This question of the paths of conduction through the auricle has been discussed already in Chapter VII.

An anatomical basis, a firm physiological basis, for sino-auricular block alike fail; the hypothesis as it is held to-day is chiefly supported by morphology. In the tortoise and in other lower vertebrates, stimulation of the vagus will produce a definite condition of block, the terminations of the veins continuing to beat while the rest of the heart remains quiescent; the tissue in this neighbourhood has apparently a relatively low conducting power which is further depressed by nervous inhibition. Eyster and Meek have reported a disorder of the dog's heart following morphia injections, a vagal disorder similar to that seen in the human subject (*159*).* If the clinical condition is shown in the future to be sino-auricular block in reality, it will probably be demonstrated that a corresponding region exists in the mammalian heart and that its function is depressed by similar influences. But that has not been shown as yet. The association of sino-auricular block with auriculo-ventricular block in the cold-blooded heart, on stimulating the vagus or when the heart is dying, is to be emphasised, especially as a similar association exists in man. If a change in the conducting power in one region of the heart is induced by a particular influence, the same influence may be expected to produce a change in conducting power in other susceptible regions.

To sum up, the hypothesis of sino-auricular block in man finds support from (1) the occurrence of a similar or closely comparable condition in the cold-blooded heart; (2) the power of the vagus to induce both the clinical and experimental phenomenon; (3) the frequent association of high level block with auriculo-ventricular block; and lastly (4) the peculiar and highly suggestive arrangement of the beats at the time of the disturbance, when this takes the form of intermissions. While support for the hypothesis is not inconsiderable, a final conclusion cannot be formed.

Amongst the difficulties hindering us from reaching a fully acceptable explanation are: (1) the occasional association of simple intermissions with more complex sinus disturbances, such as gross irregularity,† and (2) our inability to recognise an anatomical or physiological basis for a block in this region of the mammalian heart.

* The earliest record of an irregularity attributed to *S-A* block in experiment on mammals is that of Hering (*270*); but the manner in which the disturbance arose in his animals is quite obscure.

† In regard to this relation, the vagal origin of both irregularities might be regarded as a sufficient explanation.

CHAPTER XXXI.

CARDIAC SYNCOPE AND UNEXPECTED DEATH.

THE causes of sudden loss of consciousness in the human subject are manifold; in some patients a hidden or unrecognised defect in the nervous system is primarily responsible; in others the cardiovascular system is manifestly at fault. In all cases of the last group, a deficient supply of arterial blood to the brain is the direct cause of the attacks of which the patient complains; both major and minor seizures are due to cerebral anæmia.

The effects of cerebral anæmia.

In patients who suffer from heart-block, grave symptoms referable to the nervous system are not uncommon; they consist, according to the length of the attack, of pallor, transient giddiness or dimness of vision, momentary loss of consciousness, convulsions and, in the end, death. In such patients the attack is caused by profound slowing or by standstill of the ventricle. The preliminary lapse of pulse beats which, as Webster (*749*) first found, heralds the attack, the relation between the length of standstill and the qualities of the accompanying nervous phenomena, and finally the reappearance of the pulse beats some time before consciousness is restored, teach us that the loss of consciousness is secondary to ventricular slowing. The case reports afford abundant evidence of such time-relations between cardiac events on the one hand and nervous events on the other. If standstill of the ventricle lasts but two or three seconds there is little disturbance; the patient may become just aware of the cardiac default. Usually standstill for three to five seconds produces momentary loss of consciousness. If the ventricle fails to beat for longer periods, say from fifteen to twenty seconds, twitchings or convulsive movements appear, respiration deepens and becomes sighing, and the patient gradually becomes cyanosed. If the heart beat is in abeyance for ninety to one hundred and twenty seconds recovery is rarely witnessed. I give these figures after examining a number of published papers; they are, of course, approximate and variable; cerebral anæmia is tolerated differently by different patients.

Loss of consciousness and convulsive movements follow copious bleedings, whether experimental or therapeutic; and these symptoms unquestionably result from anæmia of the brain. If the carotid arteries are compressed and occluded (*404*) in young adults, dilatation of the pupil, deep and sighing respiration, dizziness and eventual loss of consciousness are witnessed. Hill (*312*) and others (*688*) produced unilateral convulsions by compressing one carotid. When we find the same symptoms associated with asystole the reason for them is clear. As soon as we are persuaded that the major symptoms of cardiac syncope are due to anæmia of the brain the first step in our study is complete. It behoves us next to inquire into the cause of this anæmia. The output of the heart is rapidly reduced either by an abrupt reduction of inflow or, the inflow remaining normal, by faulty beating. To the former group, the fainting of severe hæmorrhage belongs; the common fainting attacks which occur in emotion or in overheated or crowded rooms are often ascribed to sudden and active dilatation of the visceral blood vessels, though with insufficient reason. The subject is said to bleed into his own abdominal vessels, these suddenly relaxing to form a large potential reservoir in which the blood accumulates.* But altered distribution of the blood consequent upon vascular changes need not detain us, for it does not properly belong to our subject, as do those forms of syncope which are due to defective action of the heart.

There is as yet no agreed definition of the term "Adams-Stokes' syndrome," neither is the precise form of the fits described by Adams (*3*) and Stokes (*710*) knowable, however probable it may be that their patients suffered from heart-block. Very slow action of the ventricle is not always due to heart-block but, whatever its cause, it leads to faintness or actual loss of consciousness. Further, the pulse rate may be retarded to one-half its normal rate by frequent extrasystoles; instances of this kind are on record (*344, 690*), and one example has come under my own notice, in which epilepsy of nervous origin was accidentally associated. Thus, there are many conditions in which continued slow action of the pulse combines with fainting or epileptic attacks to form a clinical syndrome, with which the names of Adams and Stokes are often associated.

In the succeeding pages I purpose to describe a number of well-defined and distinct phenomena, each a form of disordered heart action and each known to be an occasional or frequent cause of cardiac syncope.

Forms of cardiac syncope.

1. *Standstill of the whole heart.*—Some years ago a case was reported (*572*) in which standstill of the ventricle occurred during the act of defæcation; repeated syncope occurred. The case probably belongs to the category of standstill of the whole heart, for an aneurism was found upon the basilar

* In such circumstances the attacks of faintness should be accompanied by an increased rate of the pulse.

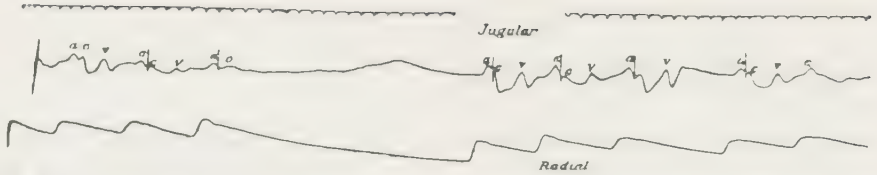


Fig. 319 (after Laslett). ($\times \frac{3}{5}$.) A polygraphic curve illustrating abrupt standstill of the whole heart as a cause of syncope.

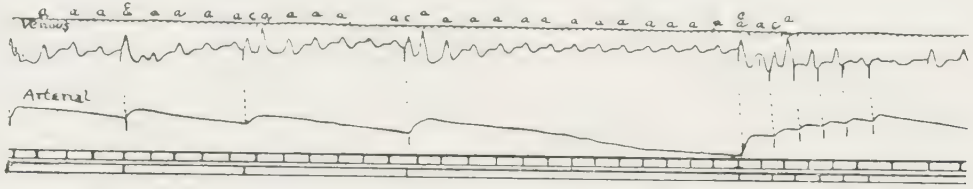


Fig. 320 (after Wilkinson). A polygraphic curve showing standstill of the ventricle in a case of chronic and complete heart-block. A somewhat unusual feature of this curve is the quickening of the ventricle following the long ventricular diastole.

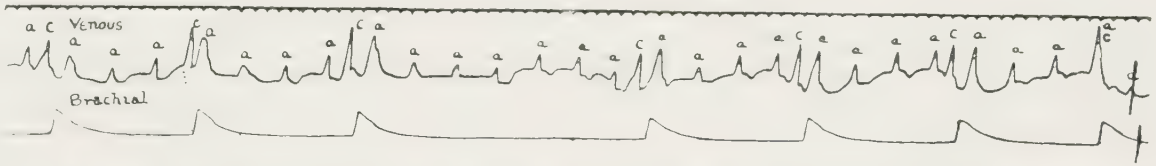


Fig. 321. ($\times \frac{3}{5}$) A polygraphic curve showing standstill of the ventricle in a case of chronic and complete heart-block.

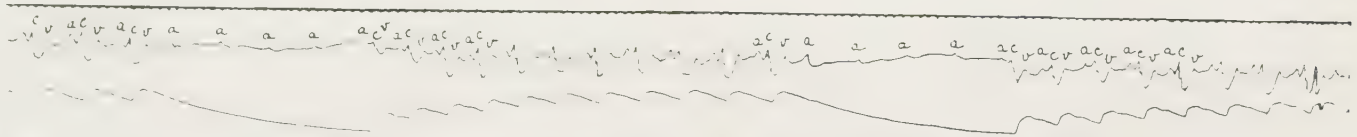


Fig. 322 ($\times \frac{1}{2}$.) A polygraphic curve showing ventricular standstill which interrupts a normal heart rhythm.

artery, so situated as to produce pressure upon the medullary centres during sudden rises of blood pressure. Unfortunately modern graphic records were not then available and there is no certain evidence, though strong presumption, of complete standstill. A case has been recorded by Gerhardt (*222, Case 4*) of a tumour compressing the left vagus nerve in which similar attacks were witnessed. Mackenzie (*515, page 345*) has published records of complete standstill of the heart in a patient who was under the influence of digitalis; pauses of two and a-half seconds occurred during which both auricle and ventricle remained quiescent. A similar case has been recorded by Wenckebach (*762*), and described by him as illustrating "Luciani's periods." A striking instance is that reported by Laslett (*409*). The patient, in whom no definite signs of cardiac disease could be found, manifested frequent standstill of the whole heart of four to eight seconds duration associated with syncope. One of Laslett's curves is reproduced in Fig. 319. In his patient the attacks were shown to be vagal in origin* for they were abolished by atropine.

2 *Slowing of the whole heart accompanied by lowered blood pressure.*— In so far as the common form of fainting attack which is provoked by emotion or by long standing has been investigated, it has been shown to be largely of vagal origin. Primarily it is not a cardiac malady but is brought about reflexly, the vagus being the chief efferent channel. A common exciting cause is the sight of blood. Such attacks are frequent in patients who are recovering from acute illness, who are chronically infected or in poor health. Presumably many of the fainting attacks of nervous women are similar in kind.† The attacks are ushered in by feelings of unsteadiness, giddiness and dimness of vision; during this period the heart's action is becoming gradually slower; at the height of the attack, pallor and sweating are prominent, the pulse is slow and the systolic blood pressure reduced‡. The pulse slowing is not very profound; the rate may fall to 50, to 40 or perhaps a little lower. The fall of pulse rate is by itself insufficient to account for loss of consciousness.§ Yet consciousness is usually lost and twitching of the body may be seen when the rate is reduced to these extents. The nervous symptoms appear when these pulse rates prevail because there is a simultaneous and independent lowering of arterial pressure; the systolic pressure falls to 60, to 50 millimetres of

* Pope's horse (*607*) which suffered from slow heart action and syncopal attacks, was found to have "dropsy of the cervical region." In Stackler's (*703*) case of bradycardia associated with syncope, the right vagus was involved in a tumour. Contractions of the *foramen magnum* and fracture of the cervical vertebræ (*36, 331*) have also been blamed as exciting causes of vagal attacks accompanied by loss of consciousness.

† The fainting attacks of aortic disease are I believe of the same nature, though it is not yet proved.

‡ Simple slowing of the heart does not lower the systolic blood pressure, the drop of systolic pressure in the patients is an added phenomenon.

§ When a fall of pulse rate is uncomplicated the fall must be usually to 20 or thereabouts before consciousness is lost.

mercury or even lower, and the pulse becomes imperceptible. Nausea commonly precedes loss of consciousness; vomiting is not infrequent. These symptoms and the rise of pulse rate which immediately follows an intravenous injection of atropine, stamp the attacks as largely vagal in origin. At all events the vagus is implicated; whether the fall of pressure is due to vagal influences, as is the fall of heart rate, is however doubtful. A full account of this form of syncope will be found in a recent paper (*79*).

3. *Standstill of the ventricle alone.*—Many instances of cardiac syncope are seen in patients who exhibit heart-block, and in these ventricular asystole is unaccompanied by a corresponding failure of auricular contraction (*15*, *21*, *143*, *200*, *351*, *625*). Such attacks are of distinct kinds:—

(*a*) *Suddenly developed heart-block.*—A few years ago I saw a patient in whom repeated attacks of cardiac syncope were witnessed but in whom *A-V* conduction was generally perfect; without warning the ventricle would cease its beating, the auricle continuing at its former rate; after a few seconds the patient would lose consciousness (*474*, *page 117*). I observed many scores of these attacks and obtained records of which Fig. 322 is an example.* Atropine failed to give relief and the man eventually developed complete heart-block and died. An autopsy was not obtained, but we may presume that the early condition was due to a lesion of the *A-V* bundle, affecting conduction in a transient and spasmodic fashion. A somewhat similar case has been reported by Cohn and myself (*69*); it was an old lady who exhibited heart-block and syncope spasmodically and the fibres of the *A-V* bundle were found widely separated by large venous sinuses. In this instance we attributed the syncopal attacks to intermittent swelling of the sinuses whereby conduction from auricle to ventricle was interrupted transiently by pressure.

(*b*) *Increase of pre-existing heart-block.*—In those who show high grades of partial heart-block, cardiac syncope is frequent. The ventricular rate is decreased during the fit, while the auricular rate is maintained or is enhanced. The retardation or standstill of the ventricle is due therefore to the increase of a pre-existing heart-block. A sudden increase in the grade of block and the establishment of 10 : 1 or 20 : 1 ratios or of complete block provides a clear solution of the ventricular slowing; such attacks are very common shortly before complete block becomes established in cases of chronic heart-block. The increased block which is responsible for the fits has been explained variously. It has been supposed that a sudden increase of auricular rate may be responsible, for, where conduction is already defective, it has this effect in experiment (*143*, *484*). In other cases the vagus† has been blamed, for fits have been provoked in these by pressing the carotid

* A very similar curve, though from a more complex case, has been published by Thayer and Peabody (*724*, *Fig. 7*), and another by Mackintosh and Falconer (*520*).

† Partial block is greatly increased by stimulation of the vagus (*484*).

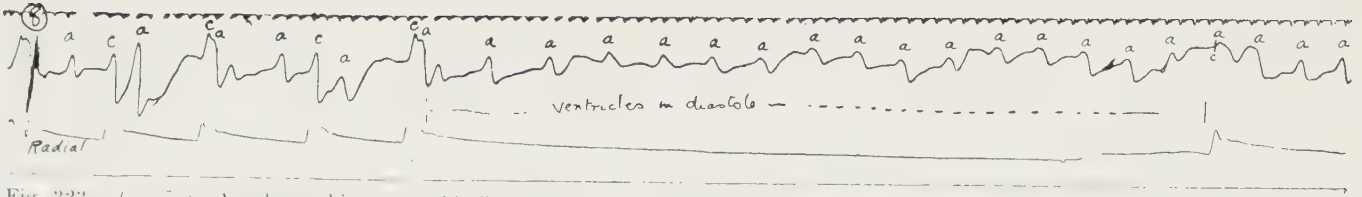


Fig. 323. (Reduced.) A polygraphic curve, kindly lent by Dr. John Hay. Taken from a case of Adams-Stokes' syndrome. The opening portions of the curve show dissociation. The ventricle suddenly ceased to beat for a period occupied by sixteen auricular cycles, a time interval of 11 seconds. The pause was accompanied by loss of consciousness, and was but one of a series of similar pauses, some shorter and some much longer, observed in the same patient on the same day.

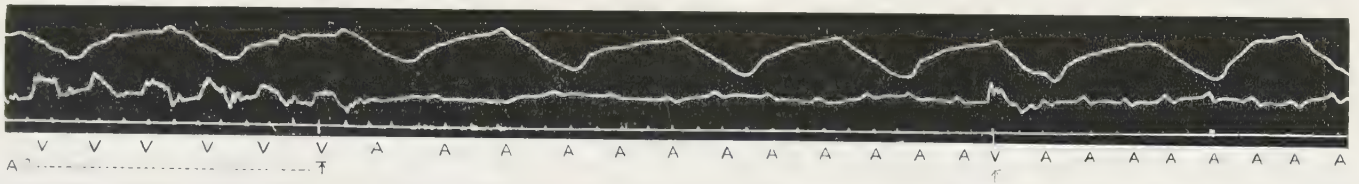


Fig. 324. (*Erlanger and Blackman, Heart, 1909-10, I, 197, Fig. 10.*) Respiratory and apical curves from a dog, five weeks after the auriculo-ventricular bundle had been crushed and its functional continuity destroyed. On the day upon which the curves were taken, the animal had syncopal attacks. The curve shows the termination of a series of rapid ventricular beats and "standstill" of the ventricle.* The auricles continue to beat. Time in fifths of a second.

* This curve is very similar to a clinical curve published by Thayer and Peabody (*724, Fig. 6*).

sheath (*625*). It is possible that transient intensification of an inflammatory process in the bundle may sometimes provoke the attacks; an increase of pressure in the blood vessels or lymph spaces of this region, by altering the tissue tensions, might abolish conduction in a bundle in which it was already precarious.

(*c*) *Ventricular standstill in complete heart-block.*—This is less frequent, though it has often been recorded (*21*, *785*) and many graphic records have been published (Fig. 320, 321 and 323).* The nature of these fits constitutes a distinct problem. The vagus has been supposed to cause the ventricular slowing. In the dog the vagus has a directly inhibitory influence upon the ventricle. The experiments (*144*, *150*, *267*, *628*) seem to show, however, that in a heart in which the auriculo-ventricular bundle has been destroyed, the vagal influence upon the ventricle is neither powerful nor invariable.† Fredericq (*190*) believes that most of the vagal fibres to the ventricle pass through the *A-V* bundle and that these are destroyed in clamping or cutting the bundle; he states that light clamping of the bundle may produce complete block while the new ventricular rhythm remains susceptible to vagal stimulation.‡ Atropine, given in full doses, has little or no influence upon the fits of complete block as a general rule, thus indicating that the vagus is not at fault. Although in one instance of complete heart-block Volhard (*743*) has been able to induce a short standstill of the ventricle by pressure upon the vagus, as a rule this procedure does not affect the ventricular rate. Upon the undisturbed idio-ventricular rhythm, atropine is usually without influence, but cases have been recorded where considerable acceleration has been witnessed (*785*) after the administration of this drug, although pressure on the vagi in the same cases failed to slow the ventricle. Briefly, it would seem that in most clinical cases of complete block, the idio-ventricular rhythm is uncontrolled by the vagus, but that in some patients (especially young patients) there is control in some degree; in these the vagus may conceivably be responsible for syncopal attacks. The presence or absence of vagal control would appear to depend, according to Fredericq's observations, upon the nature of the lesion responsible for the heart-block.

The experiments of Erlanger and Blackman (*148*) direct attention more especially to the ventricle itself. These workers have succeeded in reviving dogs after the bundle has been crushed and permanent dissociation obtained. Several of the animals subsequently developed syncopal attacks similar to

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* Cases of complete heart-block seem more prone to enter a *status epilepticus* and it is in this state that most of the curves have been taken; but cases of complete block appear to be relatively immune from seizures.

† The sympathetic on the other hand seems to possess a more striking action (*258*, *267*.)

‡ In the complete block of asphyxia the vagus acts powerfully on the rhythmic centre controlling the ventricle (*6*), but in this example we have to do with impulses arising in the *A-V* node.

those of clinical heart-block (Fig. 324), yet vagal stimulation in these animals failed to affect the ventricular rate. The fits associated with complete heart-block depend upon influences which depress the idio-ventricular rhythm, a rhythm which is known to originate in the junctional tissues (Chapter XVI).* The importance of the experimental work is twofold ; it has shown that a simple lesion of the bundle not only suffices to provoke dissociation, but also the fits due to standstill of the ventricle ; secondly, it has shown that the idio-ventricular rhythm, isolated from vagal influences, may alter profoundly, not only in its rate, but also in the sequence of its beats.

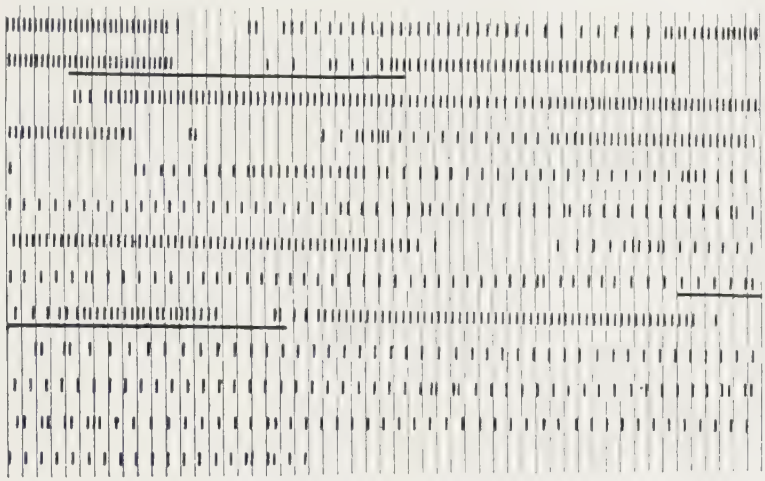

Fig. 325. (*Heart, 1912-13, IV, 18, Fig. 1.*) A diagram of the ventricular beats in a case of complete heart-block over a period during which syncopal attacks were observed. The diagram reads continuously from left to right in successive lines. Each standstill of the ventricle follows a period of enhanced ventricular rate ; the periods of increased rate being due to serial extrasystoles. The vertical lines are drawn at intervals of two seconds.

It has been shown by Erlanger and Hirschfelder (*150*), and recently their observations have been confirmed by Cushny (*90*), that after the intrinsic ventricular rhythm has become established its activity may be greatly depressed by excitation of the ventricle. If the dissociated ventricle is stimulated by successive electric shocks and its rate is artificially accelerated in this manner, standstill occurs at the termination of such stimulation, the intrinsic rhythm remaining dormant for a while and requiring some little time to develop. A perfect clinical parallel has been described by Cohn

* Erlanger (*143*) concluded that in his patient the arrest of the ventricular contractions was in some way connected with auricular acceleration, although the beating of the two chambers was dissociated. Wardrop Griffith has recorded a similar phenomenon (*236, Fig. 6 and 7*).

and myself (*73*), and more recently a second case has been recorded by Hecht (*246*). Our patient suffered from complete heart-block and fits, both of many years duration; the spontaneous rhythm of the ventricle was also disturbed from time to time by single or successive extrasystoles. Shortly before death, longer series of extrasystoles were observed; whenever such a series of faster beats ended, the ventricle remained quiescent and a syncopal or epileptic attack followed. In Fig. 325 the incidence of the ventricular beats has been plotted from curves over a period of 21 minutes. It will be observed that the sequence of the chief events is invariable. Each period of asystole is preceded by relatively rapid action of the ventricle. Thus, it has been conclusively shown that extrasystoles which temporarily enhance the ventricular rate may lead up to syncopal attacks in cases of complete heart-block, though, admittedly, syncope of this origin is unusual.

We may summarise our knowledge of the fits of heart-block by saying that in partial block they depend upon alterations of conductivity, and that in dissociation, their appearance or non-appearance is consequent upon influences which affect the pacemaker of the ventricle. In rare instances this pacemaker may be affected by vagal influences; in others, equally rare, it is affected by the interpolation of a series of extrasystoles. In the remaining cases the cause of ventricular slowing is unknown. It should not be forgotten that the pacemaker of the ventricle lies in the immediate neighbourhood of the lesion responsible for dissociation, and that in some cases this new pacemaker may be influenced by the lesion.

4. *Fibrillation of the ventricles.*—The reasons for believing this disorder to be a cause of syncope are fully set forth in Chapter XXVI. Probably, it is the chief cause of fatal syncope. Our concrete knowledge of clinical fibrillation accumulates slowly because this disorder of the heart is incompatible with continued existence.

5. *Accelerated heart action.*—When the heart of a fresh animal is excited to rapid action by interrupted stimulation, its output remains constant within wide limits of rate (*456*); there may even be some rise of mean arterial pressure; there is no fall of pressure until very high rates of beating are forced. The reserve power of the normal heart is great, and the organ accommodates itself at once to an altered rate of beating; but if the rate is greatly exaggerated, or if the muscle of the ventricle is damaged, then the blood pressure may fall rapidly and far. To illustrate the reserve capacity of the human heart, I may cite the case of a child in whom I observed a ventricular rate of 290 per minute; the observations were made while the child slept peacefully, and although the acceleration was persistent it produced no distinct signs or symptoms of circulatory embarrassment. But repeated extrasystoles (auricular or ventricular) or simple paroxysms of tachycardia are prone to induce attacks of giddiness or actual loss of consciousness in some subjects (*199, 229, 762*); in these the heart is less

tolerant ; the muscle of the ventricle, so it is judged, is affected by disease. In such, syncope is accompanied by a fall of blood pressure, the symptoms being the result of cerebral anæmia. The most notable examples of this form of cardiac syncope are seen in the case of those adults who exhibit flutter of the auricles (Chapter XXII). In flutter, syncope is a usual and repeated event and the accompanying symptoms suggest an abrupt rise of ventricular rate. Usually, the ventricular rate is one-half the auricular rate in these subjects, the former being about 300 and the latter 150 per minute ; but occasionally the ventricle responds for a short while to each auricular impulse, and assumes the full rate of 300 per minute ; such a rate is, generally speaking, not long tolerated by an adult human heart ; the arterial blood pressure quickly falls and serious symptoms manifest themselves. The curves displaying such an attack, in which the ventricle races and the blood pressure falls, were first obtained by Dr. Theodore Thompson and have been published and described by Mackenzie (*515, 3rd ed., Fig. 141*). Unexpected death in the same patients is not infrequent, and may be attributed to continued high ventricular rate.

Thus, cardiac syncope may occur when the heart's action is extremely slow or when its action is extremely fast ; each disorder compromises the blood supply to the brain. Syncope has proved in the past to be a fertile field of discussion and one in which hypothesis has outrun fact. Careful observation has taught us much that is new, but there is still a great deal to learn.

CHAPTER XXXII.

THE VAGUS.

THE vagus nerve has been the subject of numerous physiological researches (*26*, *524*) since the brothers Weber discovered its inhibitory function. Engelmann's writings conspicuously influenced current ideas of inhibitory nerve influences. Many of his observations were almost identical with those previously conducted by Gaskell, but he used them to support a different general conception. Gaskell associated function with structure, and his work went to show that the attributes of the heart musculature are possessed by different parts in different degrees and that these different parts are correspondingly controlled by the nerves. On the other hand, Engelmann's conception was of cardiac muscle fibre possessing distinct functions, each under separate nervous control. Recent observations tend in the main to uphold Gaskell's view. The sino-auricular and auriculo-ventricular nodes, formed of similar tissue elements, are structures possessing the power of elaborating impulses in a notable degree; in the dog both these nodes are under vagal control; the former is chiefly governed by the right vagus, the latter almost equally by the two vagi (see pages 164 and 191). One of the effects of vagal stimulation is to move the pacemaker from the upper to the lower end of the *S-A* node, the heart rate slowing (*483*, *551*). The *A-V* node has relatively low conducting power; its power is further depressed by vagal stimulation; in this respect the two nerves act almost equally, the left being somewhat predominant. The vagi are also believed to act upon the divisions of the bundle in the mammalian heart (see page 164). The intensive actions of the vagi, in fact the chief known actions of the inhibitory nerves, are exerted upon the special tissues. However, the vagus is also known to affect the force of contraction in the auricular and ventricular tissues of mammals; thus, while it cannot be affirmed that its action is confined to the special tissues, the conspicuous disorders of the heart beat following vagal stimulation are all due to the capacity of this nerve to govern those tissues which Gaskell would have regarded as belonging to the primitive cardiac tube.

In the present chapter we are especially concerned to note the forms of cardiac disturbance attributable to inhibitory influences, and inasmuch as these disorders have been considered in earlier chapters, the present one is necessarily synoptic.

Respiratory arrhythmias.

Respiratory arrhythmia in experiment.—In the dog and cat the lengths of the cardiac cycles vary with the two phases of respiration. With natural breathing the cycles lengthen in expiration and shorten in inspiration. The longest cycles appear when intrathoracic pressure is highest, the shortest cycles when intrathoracic pressure is lowest (Fig. 326). Section of the vagi or the injection of atropine, entirely abolishes this respiratory irregularity.

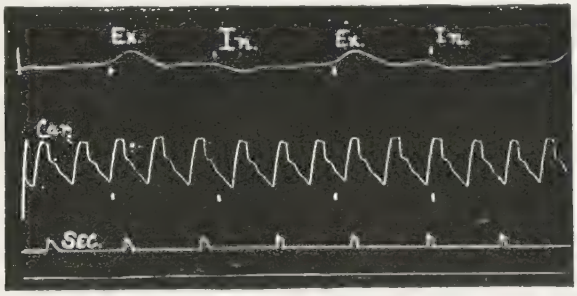

Fig. 326. (× $\frac{2}{3}$.) Simultaneous intratracheal pressure and carotid pressure curve from a cat, breathing naturally. With each normal expiration the intratracheal pressure rises, with each normal inspiration it falls. Raised intrathoracic pressure is accompanied by slowing, and lowered pressure by quickening of the heart beats. Time in seconds.

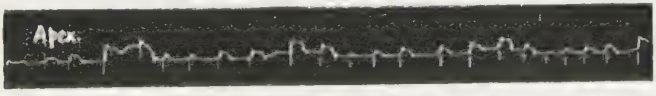

Fig. 327. (× $\frac{2}{3}$.) An apex tracing from a terrier, taken during sleep and while the breathing was slow and shallow. The periodic waxing and waning of ventricular rate is conspicuous.

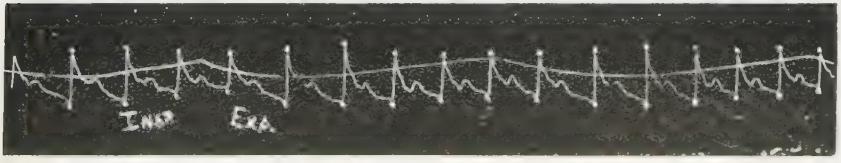

Fig. 328. (× $\frac{5}{6}$.) (*Journ. of Physiol., 1908, XXXVII, 252, Fig. 10.*) Polygraphic curve from a young adult. Simultaneous pulse and respiratory curves. There is little or no alteration of mean blood-pressure, but a conspicuous variation in the length of the pulse intervals. Lowered intrathoracic pressure (the summit of inspiration) is associated with the shortest, and raised pressure with the longest beats.

The degree of the arrhythmia which accompanies natural breathing varies much from one species of animal to another. It is very conspicuous in the dog, in which animal, during sleep and when the heart rate is slow and the breathing shallow, the irregularity is most pronounced (Fig. 327).

Respiratory arrhythmia in the healthy human subject.—In adult man, respiratory variations of pulse rate are inconspicuous or absent while the breathing is natural. But in children they are the rule rather than the exception, and constitute the chief source of irregular heart action met with in very young subjects. They are to be seen in the newly-born child.

At or about the epoch when the pulse diminishes in rate and the heart assumes the rhythm which it will maintain during adult life, respiratory irregularity may be conspicuous; occurring at the time of puberty, it has been termed by Mackenzie (*499*) the "youthful irregularity."

A periodic irregularity associated with forced breathing is universal (Fig. 328) and this arrhythmia is of the same type, and is related in the same manner to respiration as are those respiratory irregularities already described.

As has been stated, vagal inhibition is known to displace the pacemaker from the head to the tail of the sino-auricular node in certain experiments (pages 130 and 204). The same type of displacement probably happens on occasion in the human subject during deep expiration, as Wilson (*781*) has pointed out, and explains alterations in the shape of *P* in electrocardiograms during this phase of respiration. The same worker and others have noted an occasional displacement of the pacemaker to the *A-V* node in similar circumstances.

All these irregularities are known to be of vagal origin, because they are related to respiration and because they are abolished by atropine.

Respiratory arrhythmia associated with pathological processes.—While it is impossible to draw a hard and fast line between physiological and pathological phenomena, in dealing with respiratory changes of pulse rate, it may be said that these variations are peculiarly prominent in certain pathological states.

It is known that conspicuous changes of pulse rate, as accompaniments of *natural* breathing, are seen during convalescence from acute illnesses. They are also encountered in an exaggerated form in other and less readily defined conditions. Patients may be found in whom, with each act of normal expiration, the ventricular rate diminishes to one-half,* to increase once more with the succeeding inspiration (*709*) (Fig. 329 and 330).

These arrhythmias are often so pronounced in degree that they cannot escape attention. It is a rule that they are associated with reduced average pulse rate; the prominence of the arrhythmia is clearly related to the mean vagal tone prevailing at the time when the observations are made.

* They are then due, so it is suspected, to sino-auricular block.

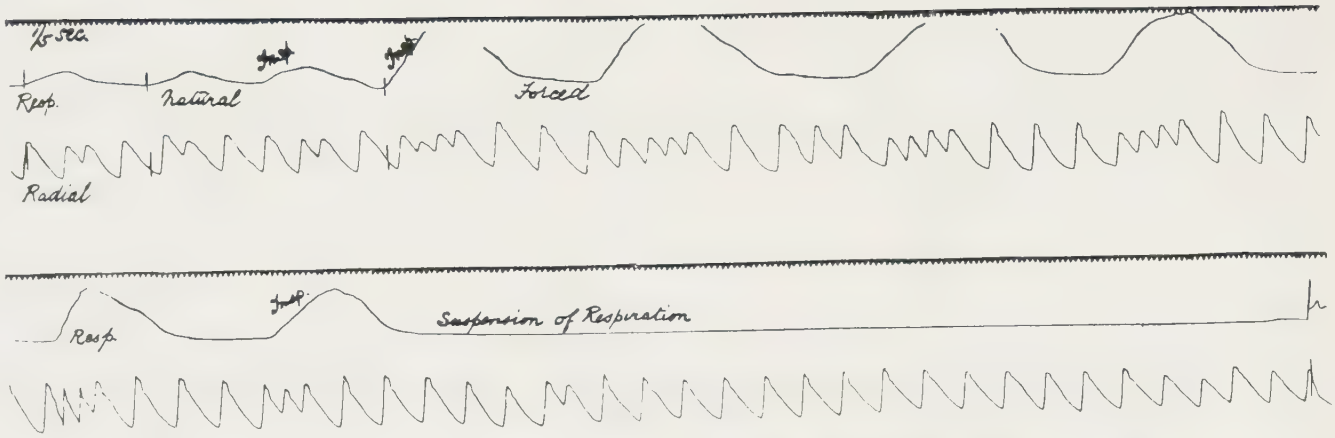

Fig. 329 and 330. (— $\frac{1}{10}$.) (*Heart, 1909-10, I, 300, Fig. 1 and 2.*) Respiratory arrhythmias observed in a case of angina pectoris. The first curve shows conspicuous quickening with natural and forced inspiration. The second curve demonstrates the almost complete dependence of the irregularity upon breathing. The pulse becomes almost regular when respiration is suspended. The whole heart was involved in the irregularity. From the same case as Fig. 315, page 349.

Respiratory irregularities affect the whole heart; each pulse cycle is accompanied in the venous curve by *a*, *c* and *v* waves, and in the electrocardiogram (Fig. 331) by natural auricular and ventricular complexes.

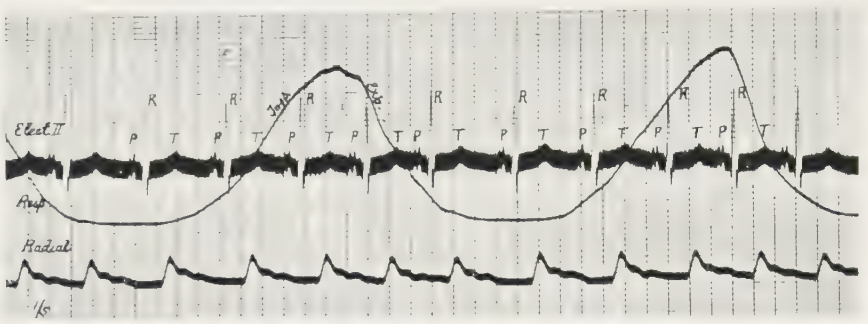

Fig. 331. ($\times \frac{3}{4}$.) Simultaneous electrocardiographic, respiratory and radial pulse curves from a child, showing a waxing and waning of heart rate with the natural acts of breathing. The irregularity affects all chambers of the heart and the normal form of electrocardiogram is maintained. Time in fifths of a second.

Disorders produced by vagal stimulation.

When the vagus is stimulated in animal experiment, the effect upon the heart rhythm is very variable. In some measure this variation is due to the strength of stimulation, in some measure it depends upon which nerve is stimulated and in some measure it depends upon individual idiosyncracy. The chief effects produced in the dog are :

1. *Slowing.*—When the excitation of the nerve is weak in intensity, the usual effect of stimulating the right nerve is slight slowing of the whole heart. The transition from faster to slower rate, and from slower to faster rate, is gradual.

2. *Standstill.*—Stronger stimulation of the right nerve usually yields standstill of the whole heart and this may or may not be succeeded by escape of the ventricle.

3. *Auriculo-ventricular block.*—This disorder is the rule when the left nerve is stimulated. As we have seen, conduction through the *A-V* node is depressed by stimulation of either nerve but is more fully displayed when, as in the case of left stimulation, the *S-A* rhythm is not materially retarded.

Compression of the vagus in the human subject.—It has been shown that inhibition of the heart may be induced in healthy subjects by firm pressure upon the neck (Czermak (*97*), Waller (*744*)). Firm pressure is exerted with the fingers directly over the carotid sheath in the middle or lower third of the neck, and the artery may or may not be obliterated before change

in the character of the heart beat is observed. The experiment is not without some slight risk. Thanhoffer (*721*) states that he took tracings from students who pressed upon their own vagi ; the compression was bilateral and serious after effects were observed in one instance. A large series of observations upon vagal compression was published by Quincke (*613*) in 1875, and recently these have been added to materially. Another procedure having a profound effect upon the heart, by a reflex path of which the vagus is the efferent channel, is ocular compression (*200*, *201*, *418*, *591-593*) ; the pressure is exerted with the finger upon the closed lids, and firmly maintained for several seconds, usually much to the discomfort of the subject. The inhibitory cardiac effects are often conspicuous.

Stimulation of the vagus in man, direct or reflex, is followed by similar disorders to those encountered in the dog. Slowing of the whole heart, standstill, heart-block, escape of the ventricle after long ventricular diastoles are the common effects in health. According to the recent observations of Robinson and Draper (*653*, see also *643*), the right nerve is more potent in producing standstill and the left in producing heart-block ; the last effect is, I think, more apparent than real.

Vagal disorders comparable to those produced by stimulation.

Phasic irregularity.—This term has been applied (*427*) to an irregularity of the human heart, in which the whole heart slows periodically, independently of respiration and without apparent reason. The phases of relatively rapid and slow heart action may be of almost equal duration (as in Fig. 83, page 137) or a natural heart rate may be maintained for several minutes and may be disturbed periodically by phases of slow action lasting some ten or fifteen seconds. The slowing and subsequent acceleration is gradual. This type of disorder is frequently associated with administration of digitalis (*655*), a drug known to exert a powerful influence upon the vagus. At other times it may occur apart from such poisoning ; it is often conspicuous in patients who are convalescing from acute fevers. It is to be seen especially well as the pulse slows after it has been accelerated by exercise.

Prolonged slowing associated with fall of blood pressure.—This form of vagal disturbance is to be distinguished from the last described. The periods of slowing are of longer duration and the intervals between them may be measured in days, weeks or months. The whole heart slows gradually and this slowing is accompanied by an independent fall of blood pressure ; the phenomena as a whole are often responsible for temporary loss of consciousness. This disturbance has been sufficiently described already on page 357.

Prolonged slow action of the whole heart.—The average rate of the normal heart in young adults at rest is in health about 70 per minute. The limits of normal rate are wide however ; instances of permanent slow action at

40 or 50 per minute are on record, and of faster action at 100 or 110, where no signs of failing health were to be observed. It is not unusual in healthy students to observe customary rates of 50 to 60.

From time to time patients are seen in whom the resting rate is as low as 30 or 40 per minute (*413*, *709*) (Fig. 332). Certain of these slow heart rates are of the nature of sino-auricular block (see Fig. 318, page 351) and these may or may not be due to the vagus ;* others are apparently of more simple vagal origin (*460*). The slow heart action accompanying jaundice is vagal,

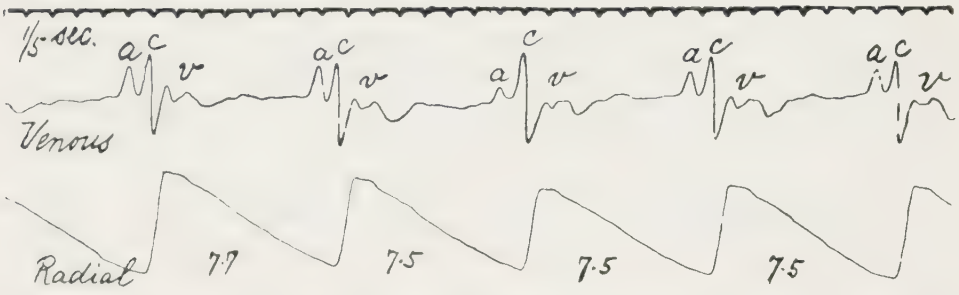

Fig. 332. (*Heart*, *1909-10*, *I*, *303*, *Fig. 6.*) Polygraphic curve showing profound slowing of the whole heart ; the rate is approximately 38 per minute. The slowing occurred in the case from which Fig. 330 was taken and is therefore attributed to the vagus.

as is that seen after recovery from acute illnesses. The bradycardia of typhoid fever is produced in another and unknown manner, for, as Marris (*540*) has pointed out, the heart in such cases is not quickened by atropine.

Standstill of the whole heart.—This is a term applied to a sudden lapse of the whole heart beat for a period of several seconds. It is fully described in the last chapter.

Auriculo-ventricular block.—The relation of the vagus to clinical and experimental heart-block is fully described in Chapters XIII, XIV and XXXI.

* The reaction of a slow heart to atropine is not the sole test of vagal origin and, as Wenckebach has pointed out, it is to be used circumspectly. Because a slow heart is quickened by atropine the slow rate is not necessarily vagal ; it may have another primary cause ; the quickening in such a case may be due simply to the removal of a normal vagal tone. The reaction to atropine should be such that the heart rate is raised from its original slow rate to the rate assumed by the normal heart under a similar dose and under similar conditions. According to Wenckebach (*765*) no bradycardia is of vagal origin in which the heart beats regularly ; this criterion is worthy of close consideration ; an association between slow action and forms of irregularity known to be of vagal type is important in distinguishing bradycardia of vagal origin. The failure of a slow-beating heart to respond to atropine does not necessarily preclude the vagal origin of slowing, for, as has been pointed out, some patients are relatively immune to atropine (*283*) ; the dose of the drug and its concomitant effect on secretion, size of pupils, etc., must be taken into account.

Sino-auricular block.—The relation of the vagus to sino-auricular block is dealt with in Chapter XXX.

Complex vagal irregularities.

Both in experiment and in man several forms of vagal disturbance may combine. Thus, slowing of the *S-A* rhythm may be accompanied by heart-block ; long ventricular diastoles are often terminated by ventricular escape. In one and the same case, an unusual respiratory arrhythmia and a phasic variation of pulse rate may be seen. An irregularity may appear which is at one time clearly associated with the acts of breathing while at other times it is independent of the respiratory acts (*709*). Very rarely, complete irregularity of the whole heart, apparently of vagal origin, may be recorded ; an example of such a disturbance is published in Fig. 333. It was taken from the same case as the curves of Fig. 330 and 332.

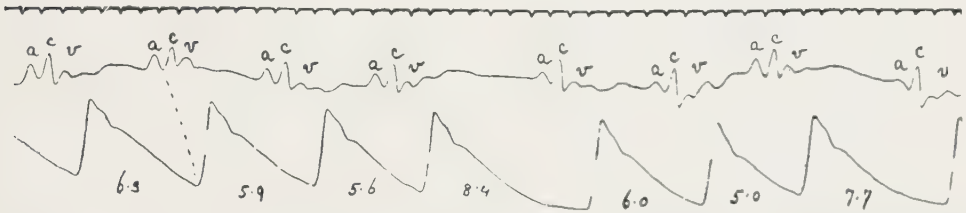

Fig. 333. ($\times \frac{5}{6}$.) (*Heart, 1909-10, I, 303, Fig. 7.*) A polygraphic curve showing gross irregularity in the lengths of pulse intervals ; the whole heart participated in the irregularity. From the same case as Fig. 300 and 302.

General remarks.

In this chapter I have attempted to bring together those disturbances of the human heart which are of proved vagal origin or which are legitimately suspected to be of this nature. In the past it has been customary to attribute many forms of cardiac irregularity and most forms of heart slowing indiscriminately to altered innervation, and these hypotheses have specially implicated the vagus. Similarly, forms of rapid heart action have been ascribed to a withdrawal of tonic vagal impulses, often upon very slender evidence. Nothnagel (*578*) believed paroxysmal tachycardia to be due to this cause.* The discovery of lesions in the *A-V* bundle producing heart-

* A full account of the vagus in relation to tachycardia in the days before graphic methods were employed will be found in Proebsting's article (*612*). Because, in exceptional cases, such as Galli's, a paroxysm is provoked by atropine, there is no reason to conclude that the paroxysm is due solely to withdrawal of vagal control ; such withdrawal may indeed be the immediately provocative factor, but it is not the basal cause (see page 347).

block and ventricular slowing, and the newly-acquired knowledge that paroxysms of tachycardia arise in ectopic foci, at once discredited and disposed of many hypotheses which were in fact almost purely imaginative. To ascribe this or that accident to the vagus or to accuse the vagus when unexpected syncope terminates life, in the absence of clear evidence that the vagus has been responsible, is not justifiable in the light of present day knowledge and modern methods of investigation.

Chapter XXXIII.

ALTERNATION.

In 1872, Traube (*729*) described under the term *pulsus alternans* a condition of the pulse in which large and small pulse beats followed each other in almost regular succession (Fig. 334). He noticed in the example described that, whereas large and small beats were placed alternately throughout, the interval separating large and small beats exceeded that separating small and large beats. The difference in the lengths of the pulse cycles may be conspicuous (Fig. 342), may be slight (Fig. 337) or may be absent. As a rule, the greater the degree of alternation, the greater is the difference in the lengths of the pulse cycles.

The variation in the height of the radial beats may be so slight as to be barely perceptible; it may be conspicuous; on rare occasions, and as a transitory phenomenon, alternate beats may be so weak that they do not appear in the pulse curves and the pulse rate is halved (Fig. 340 and 341). Alternation in the force of the pulse beats is inevitably attributed to alternation in the amount of blood thrown out by the left ventricle into the systemic arteries. The cause is clearly to be sought in the heart itself. When the heart's apex is examined its beats are found to be regular in incidence, the ventricular complexes of electrocardiograms are also placed at equal intervals* (Fig. 342 and 343).

Thus, although there is irregularity of the pulse rhythm, the ventricular rhythm is steady.

The variation in the lengths of the pulse cycles is due in part at least to variation in the presphygmic intervals; when a relatively small amount of blood is ejected from the ventricle, the rise of aortic pressure is delayed. As we have seen, a similar lengthening of the presphygmic interval accompanies extrasystoles. When extrasystoles replace each second ventricular cycle, thereby inducing a bigeminy of the heart's action, a pulse curve having a superficial resemblance to *pulsus alternans* is produced; this resemblance is naturally more remarkable and may be almost exact when the extrasystoles fall late in diastole. When the detailed analysis of pulse irregularities was in its infancy, the two types of disorder were not infrequently confused (*255*, *260*, *757*, *758*). They are usually to be distinguished

* In rare cases I have found a minute difference in the interventricular periods as measured from R to R.

by measuring the pulse cycles; for in the case of extrasystole, as opposed to alternation, the long pause follows the small pulse beat. In cardiograms of alternation the beats are quite regularly placed.

Jugular and electrocardiographic curves (Fig. 342) show the sequence of chamber contraction to be normal while the ventricle is alternating:* alternation in the output of blood from the left ventricle is due therefore to alternation in the strength of ventricular contractions.

Alternation in the amplitude of the cardiogram, alternation in the height of the chief electric deflections R and T, is often witnessed.

In man, a fully developed *pulsus alternans* is usually persistent, it does not come and go as do extrasystoles, but may be observed at each examination of the patient for weeks or even many months. In degree, however, it is variable from time to time. When less conspicuous it may disappear from moment to moment or from hour to hour. The chief factors known to induce conspicuous alternation in hearts so predisposed are an increase of heart rate and disturbance of the rhythm by extrasystoles (*513*). Alternation in the strength of the pulse beats is very frequent in paroxysms of tachycardia or flutter, where the ventricular rate is much raised. Alternation is often confined to those pulse beats which immediately succeed an extrasystole; when this happens, the first beat after the long diastole (returning beat) is large and the next beat (first restored beat) is small; there the alternation may end; on the other hand it may continue for a variable number of cycles (Fig. 335-337). This relation to extrasystoles is one of considerable interest and practical importance; the disturbance of rhythm often serves to unmask alternation when this predisposition of the heart would otherwise escape notice. Such is clinical alternation.

A precisely similar phenomenon is encountered in experiment, and the clinical and experimental findings are so completely in accord in all detail that no doubt remains that the two conditions are identical.† Amongst the earliest published examples are those used by Gaskell to illustrate his remarks in 1883 (*214*). The circumstances in which alternation appears will be discussed later.

Changes in the degree of alternation.—In hearts which are predisposed to alternate, be they human or not, an extrasystole or a slight increase of ventricular rate may bring the alternation into prominence. In other instances alternation may show itself abruptly and from time to time without apparent reason, or there may be a sudden increase or decrease in the degree of alternation which remains unexplained. These changes are well worth studying. The beginning of a period of alternation may be in a small beat

* There are rare exceptions to this statement.

† In experimental as in clinical records the beats of the ventricle are quite rhythmic. Some curves have been published of rare examples in which a variation in interventricular intervals has been observed (*325*). It is doubtful if these are legitimately to be included as examples of alternation.

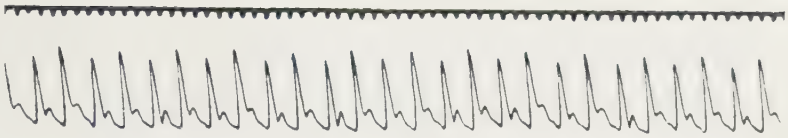

Fig. 334. An example of *pulsus alternans*. The pulse beats are alternately large and small.

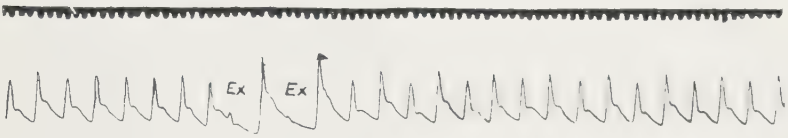

Fig. 335. Alternation in the force of the pulse beats follows a disturbance of the heart's rhythm due to extrasystoles (*Ex*).

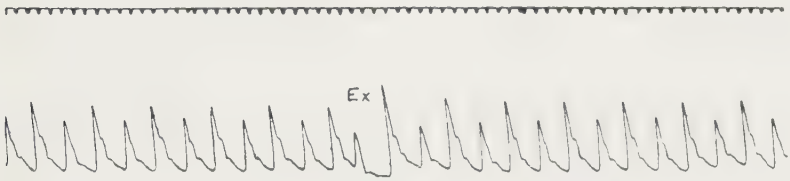

Fig. 336.

Fig. 337.

Fig. 336 and 337. Two examples showing an increase in the degree of alternation after an extrasystole (*Ex*). Note the exaggerated force of the pulse beat which immediately follows the extrasystole.

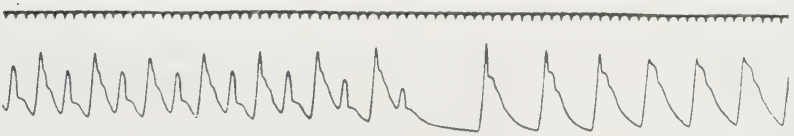

Fig. 338. The end of a period of paroxysmal tachycardia and the resumption of the normal rhythm. During the period of rapid heart action the pulse beats alternate in force.

or in a large beat. Sometimes alternation begins in a very small beat, and when this happens, the next beat is unusually large (Fig. 339); at other times alternation begins in a very large beat, and when this happens the next beat is unusually small or fails to reach the pulse (Fig. 340 and 341). As Gaskell noticed, there is a relation between the height of the large and small beats of the alternating period; what is taken from the last is added to the first. A similar statement applies to sudden changes in the degree of alternation; when a period of slight pulse alternation is continued into a period of conspicuous alternation, the change from one degree to the other is usually

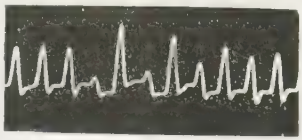

Fig. 339.

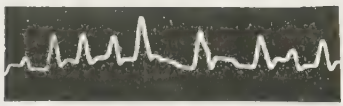

Fig. 340.

Fig. 339 and 340. Two sphygmographic curves from a patient during a paroxysm of tachycardia. Fig. 339 shows the beginning of alternation in a small beat; this is succeeded by a beat of exaggerated size. The alternation is continued but diminished, the small beats become larger and the large beats become smaller.

Fig. 340 shows alternation beginning in a beat of unusual amplitude; it continues but diminishes in degree; during this phase the small beats grow and the large beats shrink.

abrupt; at the change the tall beats grow as much as the small beats diminish. An interesting example of such changes is to be seen in Fig. 341. At the beginning of this curve (beats 6-16) the pulse is alternating slightly but distinctly; the next beat (No. 17) is the largest of the curve, it is followed by a beat of the ventricle (No. 18) which fails to reach the pulse.

It is possible that the reaction to extrasystole is not invariable; according to Windle (*787*) an extrasystole may sometimes diminish the degree of a preceding pulse alternation; but if that is so it is not the rule but a very special circumstance. It is unquestionably the rule, to which at the most there are few exceptions, that an extrasystole exaggerates the degree of a preceding alternation, or unmasks an alternation previously invisible. According to Hering the force of the extrasystolic pulse beat is itself affected (*276*). It is often unusually small. The succeeding beat is conspicuously larger than any preceding pulse beat (Fig. 335, 336 and 337).

Alternation may be brought to view by the dropping of a ventricular beat (*277*) (auriculo-ventricular heart-block) just as it may be by an extrasystole.

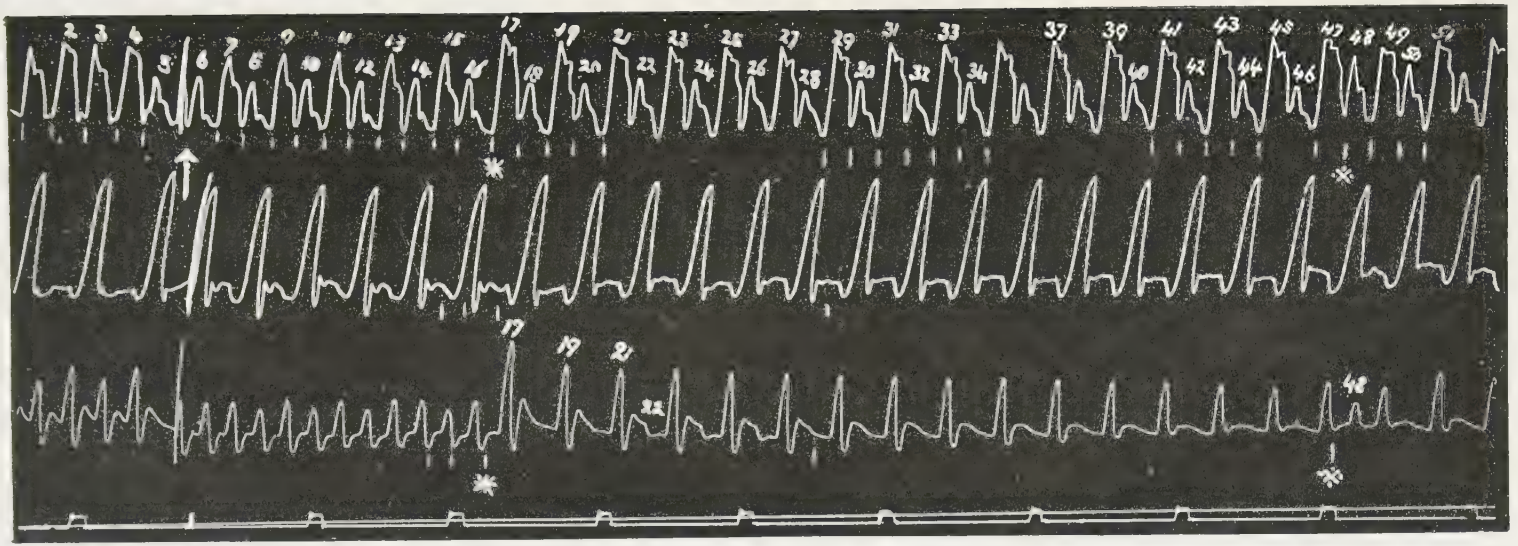

Fig. 341. Myocardiographic curves from a dog in which the right coronary artery had been tied and in which auricle (middle curve) is responding to each second beat of the ventricle (upper curve). The ventricle is alternating and interesting relations are shown between the ventricular myocardiogram and the carotid curve (bottom line). Alternation to extinction is seen following beat 17. The change starts with a large beat (17) and an accompanying rise of mean blood pressure. Note the appearance of beat 48 in the carotid curve. The time is in seconds.

Curves from the heart muscle.—If simultaneous curves are taken from the apex beat and the pulse in patients in whom the pulse shows alternation, it is the rule to find alternation in the excursion of the cardiogram. With each strong pulse beat the apex beat is strong, with each weak pulse beat the apex beat is feeble. The alternation is *concordant* in the two curves. But in other instances the curves, as Hering has pointed out (*277*, *637*), are *discordant*; the strong pulse beat corresponds to the weak apex beat. In yet other cases the apex beat shows no alternation in force while in the pulse it is plain. These observations have been confirmed (*787*). The same worker (*298*) has shown experimentally that the excursion of a myocardiographic lever recording the muscle shortening in the left ventricle may alternate concordantly or discordantly with the pulse; he has shown further that in separate records from two ventricles, or in two records from the same ventricle, discordant alternation may be observed. It is also observed that curves taken from one region of the ventricular muscle may be concordant with the pulse at one moment and discordant at another.

Electrocardiograms.—Usually in cases of slight alternation of the pulse the ventricular complexes are constant in amplitude (Fig. 343). But where the pulse shows more conspicuous change in size from beat to beat, changes are often seen in the amplitudes of the chief reflections *R* and *T* (*285*, *369*, *443*) (see Fig. 226, page 251). The amplitudes of these deflections and the amplitudes of the pulse beats may be concordant or discordant (Fig. 342). In some experimental electrocardiograms the alternate complexes show a conspicuous difference, not only in the height of *R* but in its form, and also in the height and form of *T* (*369*). These statements apply equally to the human heart.*

Circumstances in which alternation occurs.—Studied in patients, alternation of the ventricle is seen in two sets of circumstances. First, when an apparently healthy muscle is overtaxed, notably when the heart is beating at an unusually high rate. It is a frequent accompaniment of paroxysmal tachycardia. Secondly, it is found when the heart is beating within normal speed limits, but where there is reason to believe that the musculature is profoundly affected. It is particularly associated with senile changes of the heart muscle, and more especially with progressive fibrosis. It is often accompanied by grave symptoms, for example with pain of an anginal nature, and those who present continuous alternation usually succumb within a few years (*513*). It may be found temporarily in patients who are poisoned by acute infectious disease (for example pneumonia) or by digitalis (*506*).

In experiment. alternation is met with when the heart muscle is sound; thus, it is often seen when the cardiac rate is increased as the result

* Hering's electrocardiograms were taken for the most part from hearts beating very abnormally, as judged by the electrocardiograms themselves. The lettering as he applies it to his curves is scarcely to be recommended.

of repeated stimulation or where a paroxysm of tachycardia has been provoked by any means from a single point. It may be produced also by the injection of digitalis (*85*) or antiarin (*714*) (a closely allied body) into the circulation. Other poisons such as aconite (*87*), glyoxyllic acid (*369*) and hæmolytic serum

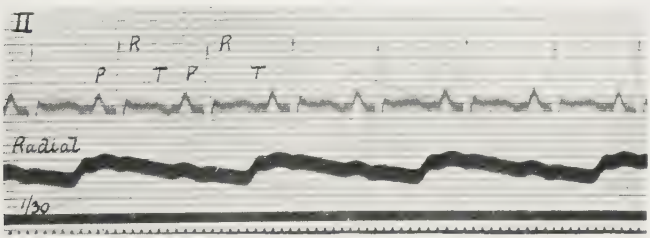

Fig. 342. Electrocardiogram and carotid curve from a dog showing alternation of the ventricle. In the pulse, alternate beats are very weak and these beats are conspicuously delayed in transmission from the heart. In the electrocardiogram the height of *R* alternates, the larger *R* corresponding to the smaller pulse beat. Produced by the injection of glyoxyllic acid. Time in thirtieths of a second.

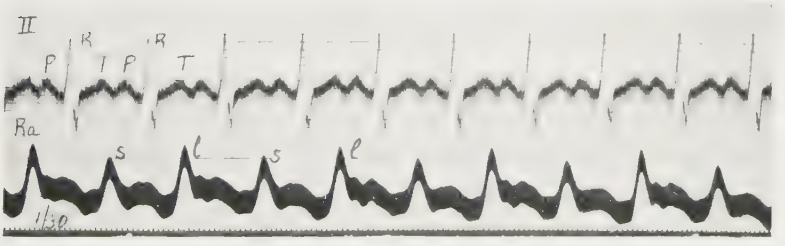

Fig. 343. Simultaneous electrocardiogram and arterial curve from a patient. The last shows conspicuous alternation; in the electrocardiogram it is not perceptible. Time in thirtieths of a second.

bring about similar results. Occurring in experiment while the heart is beating at normal rates, it indicates that the muscle is poisoned or actually dying.

Whether alternation is seen in experiment or at the bedside, there is reason to believe, either that the heart muscle has been damaged, or that a sound or relatively sound muscle is meeting an extraordinary demand for work. It is seen only when the muscle is labouring and in difficulty.

Alternation in the force of auricular contractions.

Alternation in the size of the *a* waves of the jugular pulse has often been described since it was noticed by Volhard (*742*). It accompanies alternation of the pulse, and may be concordant or discordant with it. The variation in the size of *a* waves may be due to the coincidence of auricular and ventricular systoles, alternate auricular contractions falling with the stronger and with the weaker contractions of the ventricle. As has been pointed out (*627*) the size of the *a* wave cannot be taken to measure the force of the auricular contraction; frequently, as Rihl has shown, *a* waves alternate

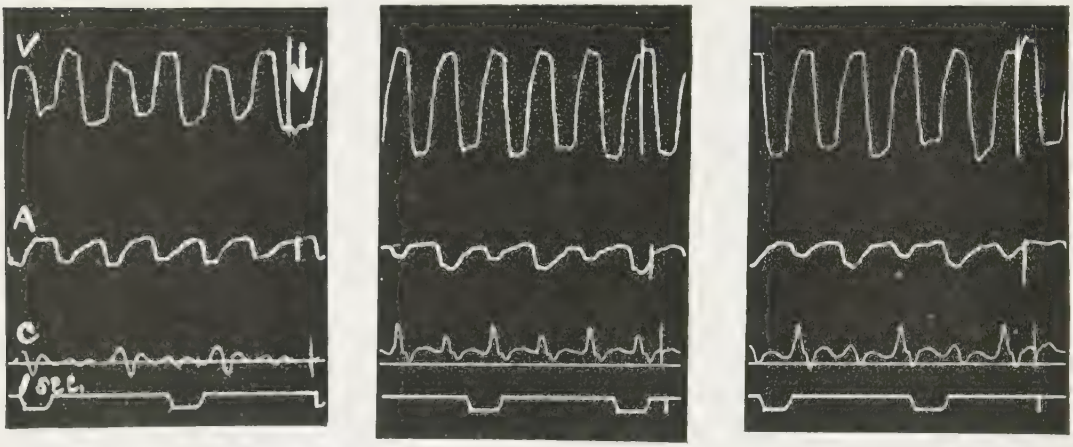

Fig. 344. (*Quart. Journ. Med., 1910-11, IV, 141, Fig. 1.*) Myocardiographic curves (*V* ventricle, *A* auricle) and carotid curve (*C*) from a dog's heart during an attack of tachycardia arising in the ventricle. The myocardiographic levers write *downwards* during systole. In the first curve the small ventricular beat corresponds to the small auricular beat and to the small carotid upstroke. In the second curve the small ventricular and auricular beats correspond to the large carotid upstroke. In the third curve the small ventricular and large auricular excursion correspond to the small carotid upstroke.

in size because they rise from different original levels of venous volume.* But that the auricular muscle may actually alternate in the force of its contractions, has been demonstrated experimentally, both for strips of the mammalian muscle (*184*), and for the muscle beating in situ (*443*). Occurring at the same time as alternation of the ventricle, the disturbances in the two chambers may be concordant (first two strips of Fig. 344) or discordant (last strip of Fig. 344).

In experiment the alternation in the *a* waves may be discordant with alternation in the excursion of a lever attached to the auricular muscle (*638*).

* For further clinical examples see Wardrop Griffith (*236*) and Gallavardin (*197, 202*).

Alternation in auricular fibrillation.

In an earlier chapter it was stated that in auricular fibrillation there is no constant relation between the amplitudes of the pulse beats and the lengths of the diastoles which precede them. That there is some relation between them is evident after even a cursory examination of a few curves. Thus, a long ventricular diastole is always followed by a large pulse beat. Where an apparent exception to this rule is witnessed, as in Fig. 348 (beat marked by asterisk), it will be found that an abortive ventricular contraction has taken place earlier. In some pulse curves from cases of auricular fibrillation the relation between pulse strength and preceding diastole is almost perfect (Fig. 345), but for the most part these are instances in which the pulse is relatively slow. Whenever the pulse is rapid the relation is broken.* The lack of relation is also shown by pulses of moderate or even slow rate in most instances. I have attributed it (*434*) to the same defect as is present in alternation of the heart. From a collection of curves it is not difficult to select examples in which, whenever a long diastole precedes a row of rapid and almost regular beats, one or more small and large beats alternate. Thus Fig. 346 displays two pauses of exceptional length and each of these pauses is succeeded by a strong pulse beat, which in turn is followed by an exceptionally weak beat (marked by asterisks). The resemblance to alternation following upon an extrasystolic disturbance of an otherwise rhythmic pulse is unmistakable. Other examples are shown in Fig. 347 and 349. Similar examples have been published by Einthoven and Korteweg (*121*), though the explanation which these writers adopt to explain it is one which it is difficult for me to accept.

The nature of alternation.

The nature of alternation has been debated at length and discussion still remains in the stage of hypothesis. For the moment, the view which holds the field is that advanced by Gaskell in 1882 to explain the curves which he obtained from the cold-blooded heart (*214*). Gaskell concluded that when the ventricle alternates the excitability of the ventricular muscle is not absolutely the same throughout, so that although the impulses which reach it remain the same in strength, yet certain parts which possess a lower excitability are able to respond only to every second impulse, while the rest of the tissue responds to every impulse. Almost all workers who have expressed their views as to the nature of alternation have supposed that the weak beat is weak because fewer muscle fibres contract; for it is agreed

* It is less apt to be broken in experiment than in patients, for in the former circumstance the heart muscle is healthy.

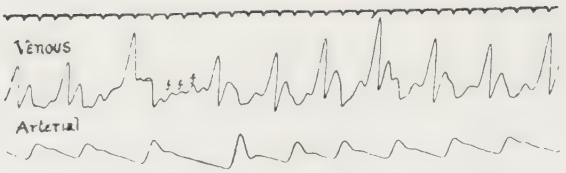

Fig. 345.

Fig. 346.

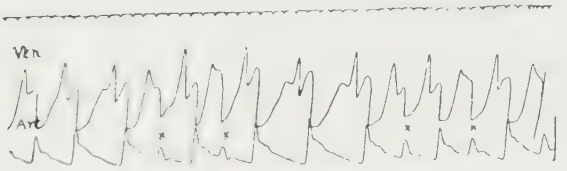

Fig. 347.

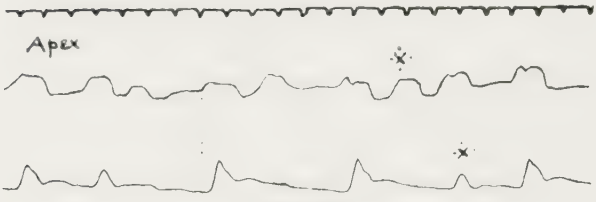

Fig. 348.

Fig. 349.

Fig. 345-349. Curves from cases of auricular fibrillation. Fig. 345.—Simultaneous venous and arterial curve; the last shows little or no sign of alternation. Fig. 346.—An arterial curve showing two beats (marked by asterisks) disproportionately small to the preceding diastoles. Fig. 347.—A similar curve accompanied by a venous tracing, showing two short phases of alternation in the force of the arterial beats. As in the last curve, the alternation appears when a series of relatively rapid beats succeeds a long diastole. Fig. 348.—A small beat (marked by asterisk) follows a long pulse cycle; this long cycle is not represented in the ventricle, for the apex curve shows an additional contraction which has failed to reach the pulse. The size of the pulse beat marked by the asterisk is not disproportionate to the ventricular diastole which precedes it. Fig. 349.—A further example of alternation of the pulse following upon a long ventricular diastole.

that if the fibres contract at all they will respond fully, according to Bowditch's "all or nothing" law; some have attributed this defect to a lowering of excitability as did Gaskell, others to a lowering of conductivity (*568*), others to a defect of contractility (*321, 329, 758*). More recently the discussion has centred around the simple failure of certain muscle fibres to contract, the extent of this failure and the disposition of the fibres concerned. The outlines of this discussion will be found in a lucid article recently contributed by Mines (*554*). He points to two views which have been held. According to the one (*276*), the contractions of the heart in alternation may be expressed by the formula V, $V-v$, V, $V-v$, where V represents the whole muscle of the ventricle and v that part of it which fails at alternate systoles. This expression of the events, which implies what Hering would term *partial asystole*, is insufficient to explain the facts and was apparently recognised by Gaskell to be insufficient. It fails to explain the increase in the strength of the large beat when alternation becomes abruptly more pronounced (see Fig. 341). The second formula proposed is one which assumes that fibres fail at each heart beat; let it be supposed that there are four heart cycles, thus :— (1) $V-v^1$, (2) $V-v^2$, (3) $V-v^1$, (4) $V-v^2$, where v^1 and v^2 represent separate groups of muscle elements but where the masses of muscle v^1 and v^2 are unequal. With an increase in the degree of alternation it is to be supposed that the failure of the muscle elements of the even cycles (2 and 4) is transferred, partially or wholly, to the odd cycles (1 and 3), thus :— (1) $V-(v^1+v^2)$, (2) V, (3) $V-(v^1+v^2)$, (4) V. According to the manner in which the transference is affected the new degree of alternation will commence with a large beat or a small one. When v^1 and v^2 contract alternately and influence equally the output of the heart, then no alternation will be displayed, but the predisposition will be present. Stated in another form, a predisposition to alternate is to be regarded as a condition of the heart in which fibres fail at each beat but in which the force of odd and even beats is nevertheless equal; such a condition may be termed, as it has been termed (*87, 329*), a hypodynamic condition.*

Suppose now that a heart in this state is disturbed by an extrasystole. When this extrasystole occurs, because of its prematurity and the short period for the recovery of the deficient fibres, neither of the deficient groups of fibres will contract and the beat will be exceptionally weak. The long diastole follows and provides ample additional time for recuperation. With the first beat of the returning rhythm both groups of deficient fibres will respond and the beat will consequently be exceptionally strong. At the next systole a few deficient fibres will have recovered, but the majority will not be ready and consequently the beat will be weak. At the next systole the majority, being rested, will contract. So the deficient fibres will

* Hering (*300*) now seems to prefer the term hyposystole for this condition; this term is open to the objection that it implies a *weak* contraction of individual fibres; Cushny (*87*) appears to use the term hypodynamic to express diminished excitability.

rearrange themselves into two groups, the preponderant number being for a greater or lesser period in the alternate beats of the restored rhythm. The hypothesis as thus held may be expressed incompletely as follows :—

Initial cycles.	Extrasystole.	Returning beat.	1st restored cycle.
$V-v^1$, $V-v^2$, $V-v^1$, $V-v^2$,	$V-(v^1+v^2)$.	V	$V-(v^1+v^2)$.

The curious example of discordance between pulse curves and myocardiographic curves is explained by supposing that the lever attached to the ventricle is especially affected by that group of muscle elements which fails for the series of ventricular beats which are relatively efficient in expelling blood (say the odd series), while the more important group which fails at the even series of beats does not include many of the fibres which pull upon the recording lever. Similarly, discordance between alternation of the pulse and apex beat is explained on the assumption that the group of muscle fibres which fails at the relatively strong ventricular contraction is a group which particularly influences the apex beat, while the larger group of deficient fibres, failing at the weak ventricular contractions, exerts a lesser influence upon the apex beat.

It has been argued by Fredericq (*184*) that this hypothesis of partial asystole is untenable, in that alternation may be observed in isolated strips of muscle which visibly contract from end to end at each beat. He would assert, therefore, that no part of the strip fails to contract. This counter argument tacitly assumes that the muscle fibres of the group which is supposed to fail lie together, constituting a particular region of the strip or of the heart wall. Such an assumption is not to be supported ; if failure to contract occurs it is as probable, if not more probable, that fibres are implicated in all regions of the heart, but that they are diffused throughout the muscle ; briefly, it is assumed that all regions of the muscle contain defective fibres, that there is a greater concentration of them in one region than in another. The hypothesis, stated in this fashion, is consistent with our knowledge of alternation as it has so far been considered and would explain those minor variations in the ventricular complex of the electrocardiogram,* which are seen from beat to beat, while the general physiological type of electric curve is maintained throughout ; for the order in which the muscle elements are activitated would not be disturbed in these circumstances.† The hypothesis is also consistent with the relation of alternation to rapid heart action, for rapid action reduces the period of rest enjoyed by the fibres and tends to delay recovery from one systole beyond

* And also the more profound changes sometimes encountered, see Kahn's curves (*369*)

† The change and variations in the electrocardiographic curves of axial leads are too small, so it seems to me, to be compatible with the failure of relatively large and solid masses of the ventricular substance.

the instant when the succeeding beat becomes due. It is in harmony with the observed influences of vagal and sympathetic stimulation upon alternation (*183*, *300*). Independently of changes of heart rate, an increase of vagal tone increases, whereas an increase of sympathetic tone decreases the degree of alternation (*299*); the first influence delays, the second expedites the recovery of the muscle functions.*

Some recent facts brought to light by Straub (*712*), however, are not easily explained by the hypothesis that has been under discussion. This worker has inscribed curves of the alternating ventricles and finds that the rate at which the heart volume increases and the extent of diastole after the large beat is much less than after the small one. In other words, the ventricles contain less blood at the moment when the weak systole begins than when the strong systole begins. It looks therefore as if the weak beat as expressed in the pulse, is not to be ascribed—at all events not entirely—to deficient contraction but to the incomplete diastole which precedes it. In instances where alternate beats are almost extinguished, the small beat takes place at a time when the ventricle, in so far as its volume and content is concerned, is still in a position of systole. The slow filling at alternate diastoles is due according to Straub to the slowness with which intra-ventricular pressure falls in diastole, and this is due to some peculiarity, at present imperfectly understood, of the ventricular relaxation. The only way, so it seems to me, in which the hypothesis may be harmonised with these facts is by supposing that the more powerful systole is unduly prolonged, that the contraction of certain muscle elements is delayed while other fibres are relaxing, thus rendering the early parts of diastole incomplete and imperfect. This may or may not be the explanation.

* Mines states that vago-sympathetic stimulation in the frog and bathing the heart with Ringer's solution, both of which procedures raise the excitability of the muscle, remove alternation. Excitability is especially suspected to be at fault. Cushny (*87*) found excitability reduced while the ventricle alternated.

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Since Ángyán's observations were undertaken in my laboratory, we have found that the rhythm of the ventricle in asphyxia emanates from the *A-V* node: his observations are to be interpreted, therefore, as effects on *A-V* rhythm and not on idio-ventricular rhythm.

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